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THE ESSENTIAL PATHOLOGY FOUND IN DIPHTHERIA, SCARLET FEVER AND TUBERCULOSIS*

With Special Reference to Involvement of the Heart Based on Review of Necropsy Cases at the Boston City Hospital for the Past Forty Years

Illustrated By Lantern Slides

BY RALPH L. IRWIN, M.D.†

DIPHTHERIA

IN diphtheria, the bacillus remains in the local lesion which is usually in the upper respiratory tract. The organism secretes a toxin which produces the visceral lesions.

This picture is often complicated by the presence of other organisms, such as the streptococci or staphylococci, which are frequently found in the blood stream and viscera in this disease.

The essential pathology found in cases of diphtheria consists of the following: (1) The local lesion. (2) Neuritis. The nerves to the palate, to the external rotators of the eye, and the vagus are most frequently involved. Peripheral neuritis is usual, and death of the nerve cells very rare. (3) Cardiac lesions. Either the myocardium, the conduction bundle, or the nerves supplying the heart may be involved. (4) Nephritis occurs occasionally. While it is usually interstitial or tubular, it may be of the glomerular type. Even with severe symptoms, this renal involvement is rarely sufficient to cause death. Complete repair of renal lesion usually occurs.

The causes of death in diphtheria are as follows: (1) Extension of membrane in about one-fourth of cases, (2) Bronchopneumonia, (3) Toxemia and septicemia, and (4) Cardiac involvement in approximately one-fifth of cases.

Involvement of the Heart:

The important cardiac lesion is myocarditis. It may be focal or diffuse. In myocarditis the muscle shows fatty degeneration, hyaline degeneration, or necrosis in a large percentage of cases. Muscle tissue once destroyed does not regenerate.

Endocarditis and pericarditis (usually due to streptococci) occur in less than 2 per cent of cases.

Insufficient studies have been made on lesions of the conducting bundle to evaluate their significance.

SCARLET FEVER

An exotoxin is also secreted in scarlet fever. A hemolytic streptococcus is nearly always found in the throat, and often in the blood stream and viscera of these patients.

In scarlet fever patients necropsied, the most frequent findings are: (1) Bronchopneumonia, (2) Pleurisy and pleural effusion, (3) Otitis media, (4) Nephritis. This last is the least common of the complications. It is usually interstitial or tubular, but may be of the glomerular type. Death from uremia occasionally occurs. Chronic lesions occur not uncommonly. (5) Cardiac lesions are uncommon. They include pericarditis which is usually associated with pleuritis, streptococcus endocarditis in less than two per cent of cases and, very rarely, myocarditis, either of the toxic or infectious types. (6) Occasional findings are ascites, peritonitis, lung abscess, abscesses elsewhere especially following trauma.

TUBERCULOSIS

The cardiac lesions in tuberculosis are pericarditis and, rarely, miliary involvement of the myocardium. Congenital malformations are said to predispose to cardiac involvement.

*Read at the meeting of the New England Heart Association, Cardiac Course, in Boston, March 2, 1932.

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HEART IN DIPHTHERIA AND SCARLET FEVER*

BY EDWIN H. PLACE, M.D.†

THIS paper is not planned to set forth a complete presentation of the problem of heart disease in diphtheria and scarlet fever but to present a concise brief picture of the disease from work now being conducted by Dr. James Faulkner, Dr. W. R. Ohler and the reader, which is still incomplete.

The heart damage in diphtheria is due to the action of toxin on the muscle and the conducting mechanism. Endocarditis and pericarditis are very rare, and when they occur are clearly related to other complicating infections. The frequency of heart involvement is 3 to 5 per cent in our cases, averaging 4.3 per cent in the past five years. There is very little difference in the incidence at different ages.

Factors leading to diphtheritic carditis are severity of disease, delay in antitoxic treatment, and, to some extent, the location of the diphtheritic infection. A great majority of cases in which heart injury occurs are those of such severity that the probability of such complication can be foreseen. Occasionally, however, due to some peculiar susceptibility, heart damage may occur in very mild cases. The average time at which these heart cases have come under antitoxin treatment is about four days. In no case have such patients been treated during the first day. The faucial and nasal vault locations are the chief sources from which these toxic damages arise. In rare cases diphtheritic infection of wounds or extensive involvement of the skin has led to diphtheritic myocarditis. In no cases of location solely in the larynx or below, in our series, has any heart injury resulted.

The onset of diphtheritic carditis is from the sixth to the twenty-first day after the beginning of diphtheria. As a rule the earlier the onset, the graver the prognosis. Diphtheritic paralysis, especially of the throat, practically always precedes or accompanies the heart damage.

Symptoms usually appear suddenly. Pallor, lassitude, nausea, vomiting, epigastric pain and restlessness may be present. Cyanosis, dyspnea, orthopnea and edema are almost invariably absent. There is no pulmonary congestion.

Physical findings besides pallor and coolness of extremities and the skin include changes in the heart sounds and the heart rhythm. The first change is the softening and shortening of the first sound. The diastolic pause is usually shortened. Rhythmic disturbance consists most commonly of gallop type of three or four sounds, rarely flutter or fibrillation. The pulse may be markedly slow, although this is rare, normal or rapid, the average about 120. The heart area may be normal or considerably enlarged. The cardiac impulse is weakened, diffused, or lost. Tenderness in the right hypochondrium and epigastrium with enlargement of the liver is common in the severest cases. The blood pressure falls.

The electrocardiograph is the only means of determining the exact nature of the disturbance of the mechanism. In no case, however, in our wards has the electrocardiograph demonstrated heart damage which was not also evident from the clinical examination. In a few cases clinical recognition of the condition was preceded by two days electrocardiographic evidence. In some cases, however, the electrocardiograph has demonstrated a high degree of heart block where clinical evidence entirely failed to suggest its presence, although showing definite heart injury.

Electrocardiograms show in the main two types of damage: First, auriculoventricular and intraventricular block, and secondly, changes in the shape and direction of the T wave and reduction of the voltage of the heart currents. These two changes may be combined, or the latter may occur alone. The cases, in which no heart block appears but in which the electrocardiogram shows myocarditis, apparently invariably recover under proper treatment. Patients presenting heart block show a very high mortality, although in the less severe blocks, recovery occurs. We have not been able to distinguish with any certainty the cases in which heart block is present from those with the marked myocarditis without heart block, except by electrocardiograph.

One of the striking facts of diphtheritic myocarditis is the disproportion between the clinical evidence in examination of the patients and the seriousness of the cardiac damage as demonstrated by electrocardiograph. Patients may seem to be in extremely good condition with relatively slight cardiac disturbance when the electrocardiograph will show such a high degree of block as to be incompatible with recovery. In these cases death often comes without any warning.

Sudden death has classically been associated with the early convalescence of diphtheria. This frequently comes like a bolt from a clear sky. In none of our cases, however, has cardiac death occurred without clear evidence of involvement preceding death. Death may occur within twenty-four hours after the onset of appreciable heart damage. The average time is about three days. Fatal cases with very rare exceptions die before the end of the fourteenth day of the

*Read at a meeting of the New England Heart Association, Boston, Massachusetts, March 2, 1932.

†Place—Clinical Professor of Pediatrics, Tufts College Medical School. For record and address of author see "This Week's Issue," page 900.

diphtheritic infection. In no case in the past five years in which the history could be accepted as reliable has death occurred from this cause alone after the twenty-first day of the diphtheritic infection. In the cases which survive, clinical recovery occurs in about a month, although electrocardiograph changes may be demonstrated for a period as long as one year.

The mortality of diphtheritic heart damage is between 50 and 60 per cent. The highest death rate is between one and two years of age, where it is close to 100 per cent. From two to ten years it is about 50 per cent, and in the adult cases in our series about 25 per cent.

Treatment of diphtheritic heart damage is very unsatisfactory. No means of definitely modifying the course of diphtheritic injury can at present be accepted. During the past year intravenous 50 per cent glucose solutions have been used routinely, and occasionally previously to that. Electrocardiograms taken before, during, and after the use of glucose have shown no striking influence on the cardiac action. There has been a very slight increase in the voltage of the heart currents and in one case sino-auricular control was restored following the glucose ingestion. In this case, however, there were several alternations between the auriculoventricular pacemaker and the sinus node so that it may have been a coincidence. In no instance was there convincing evidence that the natural course of the disease was modified to an appreciable extent.

The essential treatment is rest of the highest degree. This requires horizontal position, the securing of the utmost quiet possible, the use of sedatives, such as codeine, sodium-luminal, the marked reduction of food by mouth and if vomiting, complete omission of food. Supply of fluid may be kept up by glucose solution by rectum, and by intravenous injections.

The fact that heart injury usually causes death within one to two weeks, or that gradual and often rapid improvement occurs ending in complete recovery, gives hope that if the demand on the patient's circulation can be kept below the remaining cardiac capacity for a relatively short time, the patient will pull through. We have not yet found evidence of persisting circulatory damage in any of the patients who have recovered, although a complete check-up of a large series has not yet been finished.

Prevention of the heart injury is entirely feasible. In over 700 cases of diphtheria occurring under observation in the hospital among the employees, no fatality has occurred in the last thirty-five years. No case of cardiac damage has appeared in patients receiving antitoxin treatment during the first twenty-four hours and only rarely when antitoxin was used during the second twenty-four hours. Prevention of diphtheria by active immunization of course excludes this danger by striking at the root of the trouble.

The accompanying electrocardiograms show some of the numerous changes in the disease with brief clinical histories.

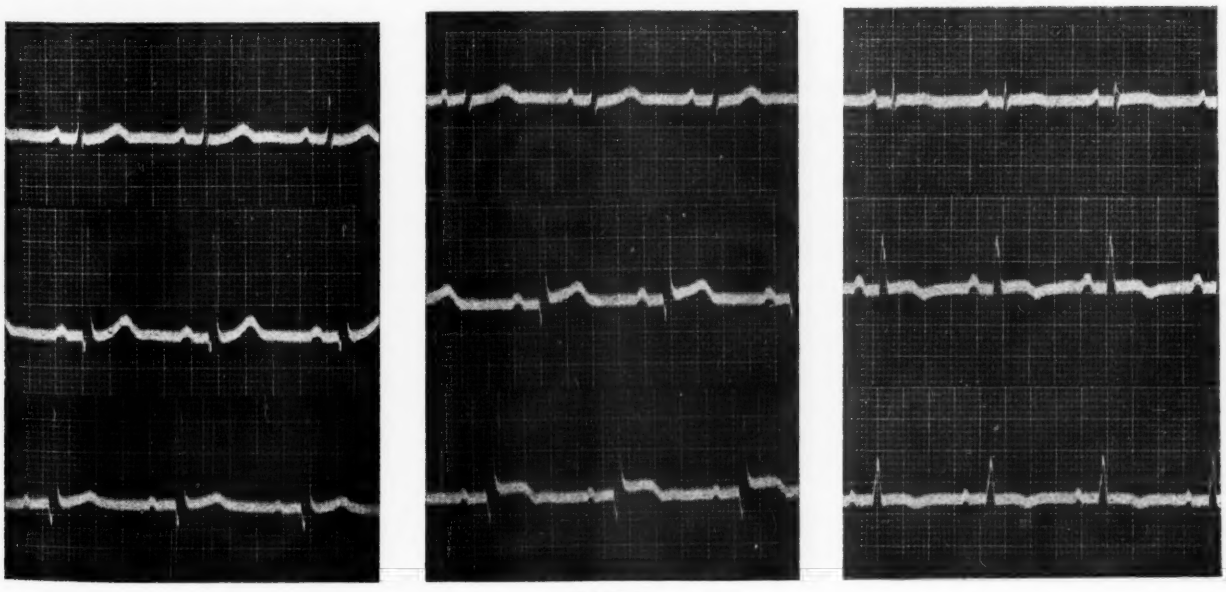
ELECTROCARDIOGRAMS

CASE 1. Plates I, II, III and IV.

H. S., aged 26 years, admitted April 22, 1931, on fifth day of faucial diphtheria. Marked swelling of throat with thin membrane covering the tonsils and right pillars. T. 100.6°. 60,000 units antitoxin were given intravenously on admission. Cardiac onset 11 days after onset with pallor, weak first heart sound. Vomiting on third day and fourth day. General improvement on the sixth day of the cardiac symptoms. In two weeks, cardiac findings were normal, clinically.

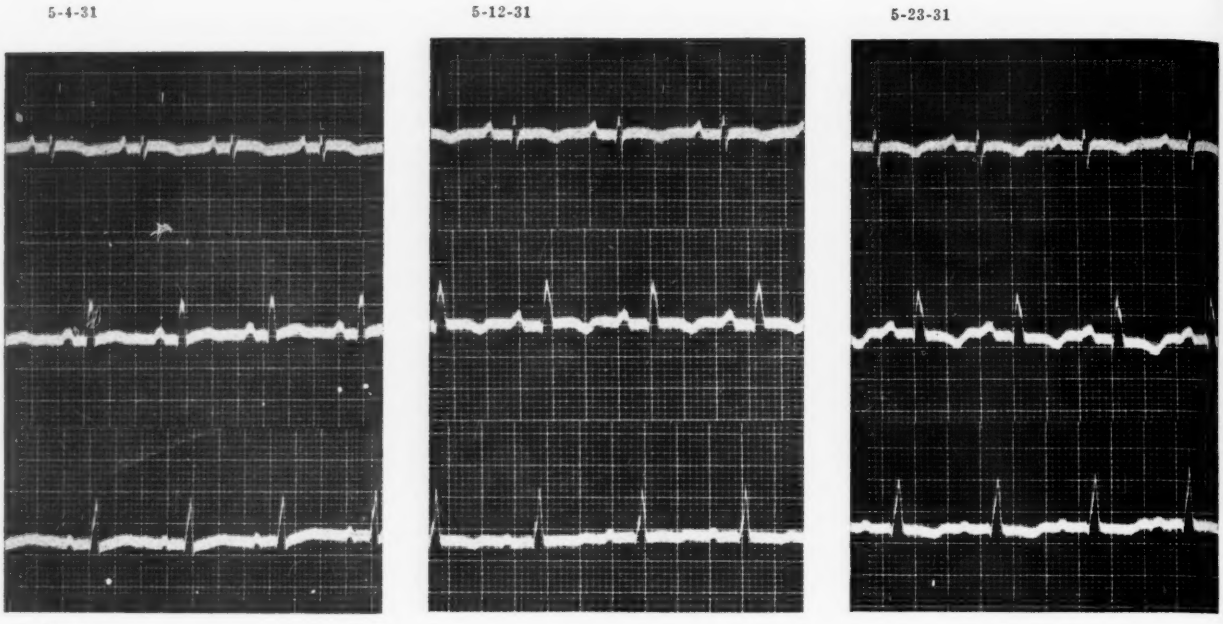
Blood pressure	
4-23-31	114-84
4-28-31	102-64
5- 4-31	80-40
5-13-31	116-76

4-29-31 4-30-31 5-2-31



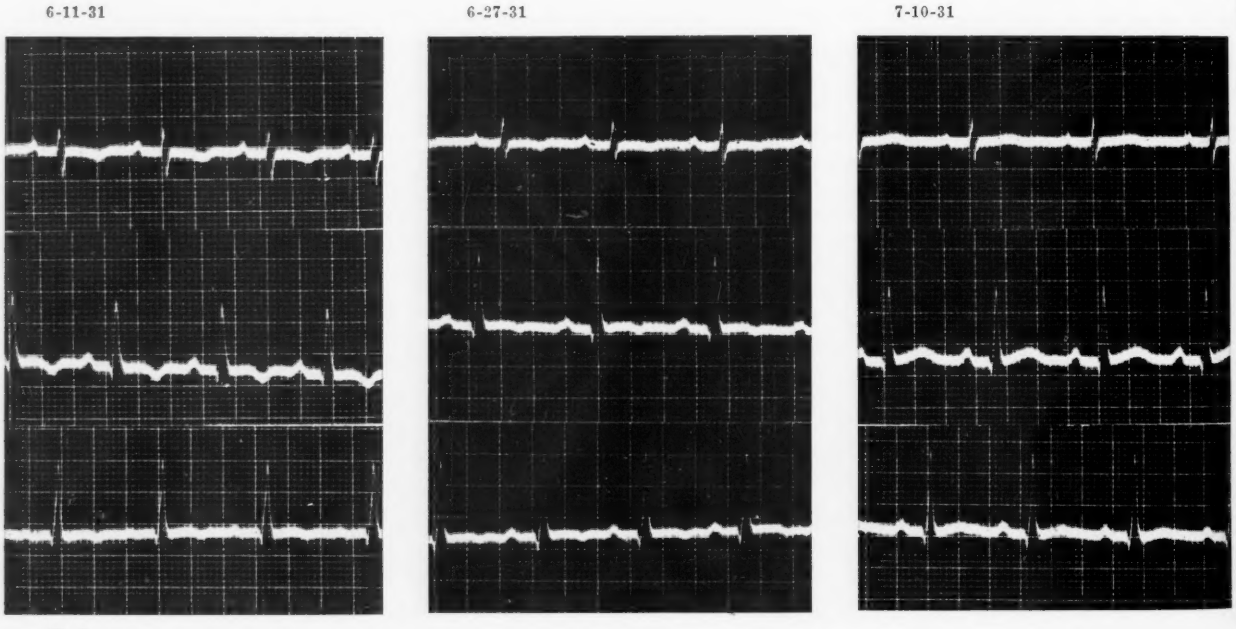
H. S.

PLATE I.



H. S.

PLATE II.



H. S.

PLATE III.

Paralysis appeared in throat on April 28, 1931 and included ciliary muscles, swallowing muscles and the extremities slightly. Considerable sensory disturbance (numbness, tingling, et cetera), of face, hands and feet. Paralysis was essentially gone in three months. Discharged July 31, 1931.

Electrocardiograms, of which 11 are shown, demonstrate:

(1) a steady drop in the amplitude of the Q.R.S. from 1.3 millivolts to 0.65 millivolts on the seventh day after onset of heart symptoms and then a gradual rise in the voltage to 1.5 millivolts in ten months,

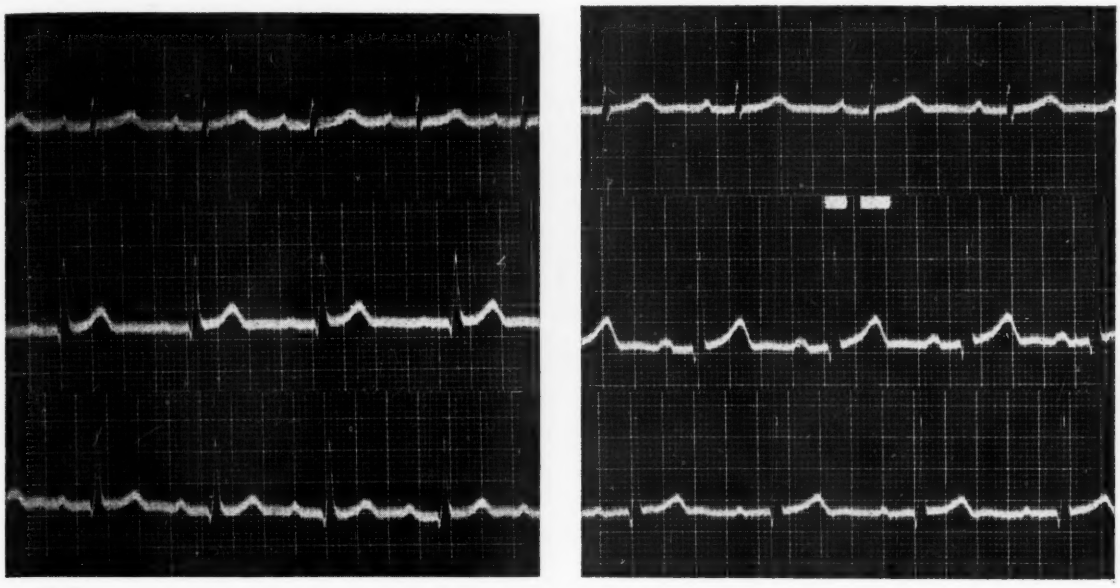
(2) a rapid inversion of the T waves, with the progress of the myocarditis, of the character seen in coronary thrombosis and a gradual return to the upright position in about ten weeks.

(3) right axis deviation,

(4) slightly prolonged PR interval, at the onset to 0.21 sec., decreased to 0.18 during early stage (seventh day of heart symptoms) and again becoming prolonged to 0.22 sec. at the end of 10 months when he was working.

In this case the chief damage appears to have been to the muscle, the conducting system almost wholly escaping.

11-12-31 1-23-32



H. S.

PLATE IV.

CASE 2. Plates V and VI.

R. A., aged 6 years, admitted April 8, 1931. Moderately severe faucial diphtheria, with swelling of throat and thick membrane covering tonsils, pillars, parts of uvula and soft palate, said to be of two days' duration.

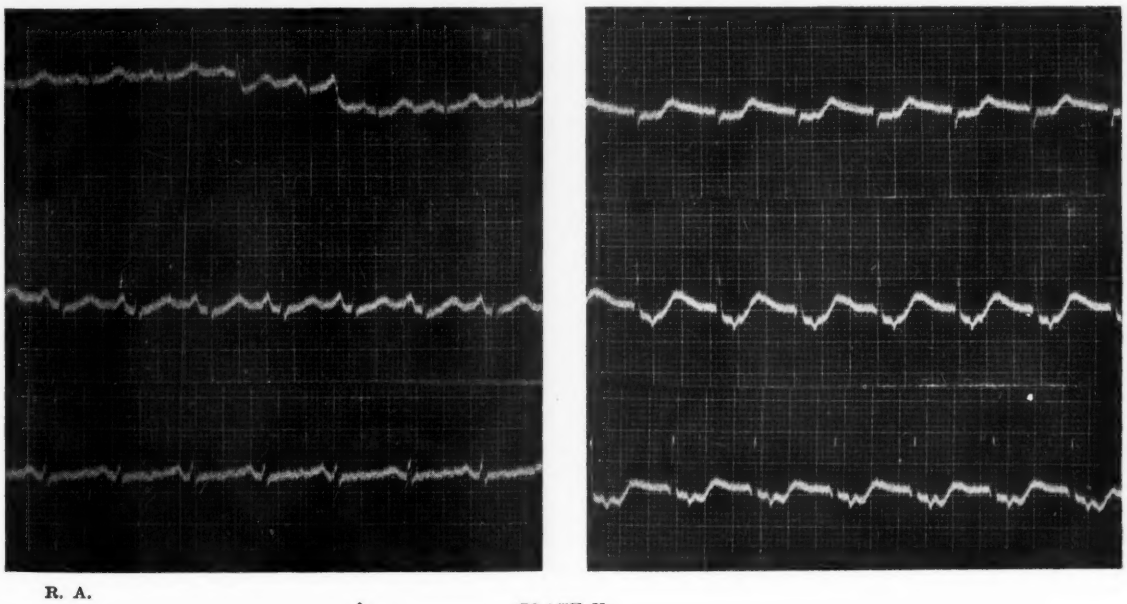
Fifty thousand antitoxin units given intravenously on admission. Throat clear in five days. Cardiac onset April 12, 1931, repeated vomiting, color pasty. Heart sounds of poor quality. Third heart sound. No epigastric tenderness, liver at costal margin. Spleen not felt. Vomiting continued at intervals, but patient not restless or in distress. Rhythm varied from a three sound gallop to absolute irregularity.

Blood pressure	
4-10-31	82-48
4-13-31	70-38
4-14-31	74-50
4-16-31	70-48
4-17-31	66-48

Died April 18, 1931.

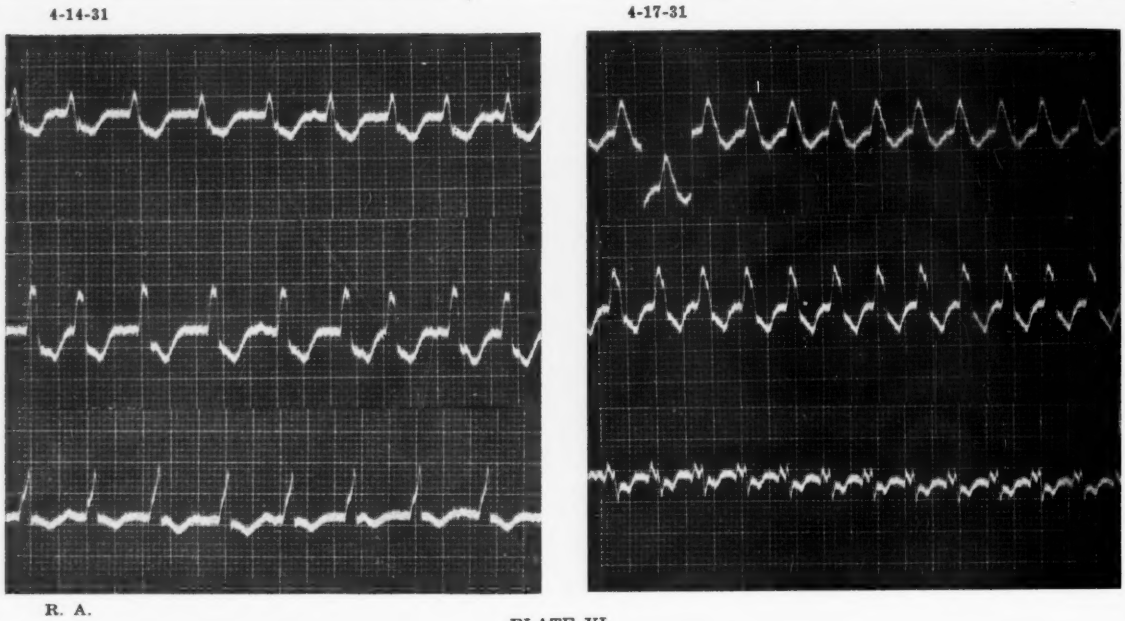
Electrocardiograms showed a change first to AV nodal rhythm with retrograde P waves, the RP interval being the same as the previous PR interval, and soon a complete AV dissociation with an auricular rate varying from 85 to 115 and a ventricular rate varying from 120 to 200, with inverted T waves. The Q.R.S. interval varied from 0.9 to 0.11 sec., and the R was slurred. Just before death the heart rate became very rapid. This patient had a severe damage of the conducting mechanism with AV block and intraventricular block.

4-10-31 4-12-31



R. A.

PLATE V.



R. A.

PLATE VI.

CASE 3. Plates VII and VIII.

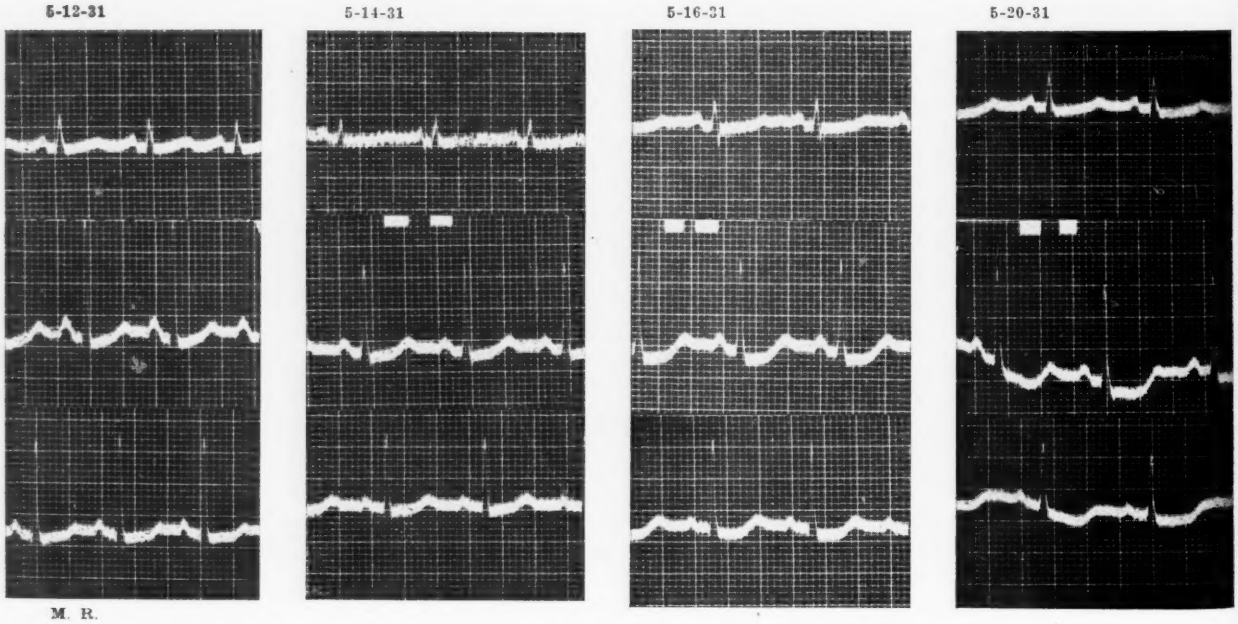
M. R., aged 9 years, admitted April 11, 1931 from the accident floor where an emergency tracheotomy had to be performed to relieve severe asphyxia from the throat swelling. Extensive swelling of throat, with membrane covering over fauces and soft palate and filling both nostrils. Neck much swollen. Duration four days. One hundred thousand antitoxin units given intravenously on admission. Membrane extended for one day and was practically gone by the fifth day after admission. Trachea tube out on seventh day. Cardiac onset on eighth day after onset with softening of the first sound. May 22, 1931, epigastric pain, vomiting, pallor appeared. Left heart border outside nipple line. Liver just felt at costal margin. These symptoms decreased during the following week. Vomiting did not recur. Paralysis of the palate on the eleventh day followed by ciliary paralysis and sensory paralysis at the end of two months. The paralysis was essentially absent at the end of three months.

Blood pressure

4-12-31	118-68	4-25-31	72-16
4-14-31	110-60	4-26-31	72-16
4-16-31	104-50	4-28-31	82-30
4-18-31	76-34	5- 1-31	82-20
4-21-31	68-20	5- 4-31	94-40
4-22-31	66-14	5- 8-31	108-55
		5-12-31	112-62

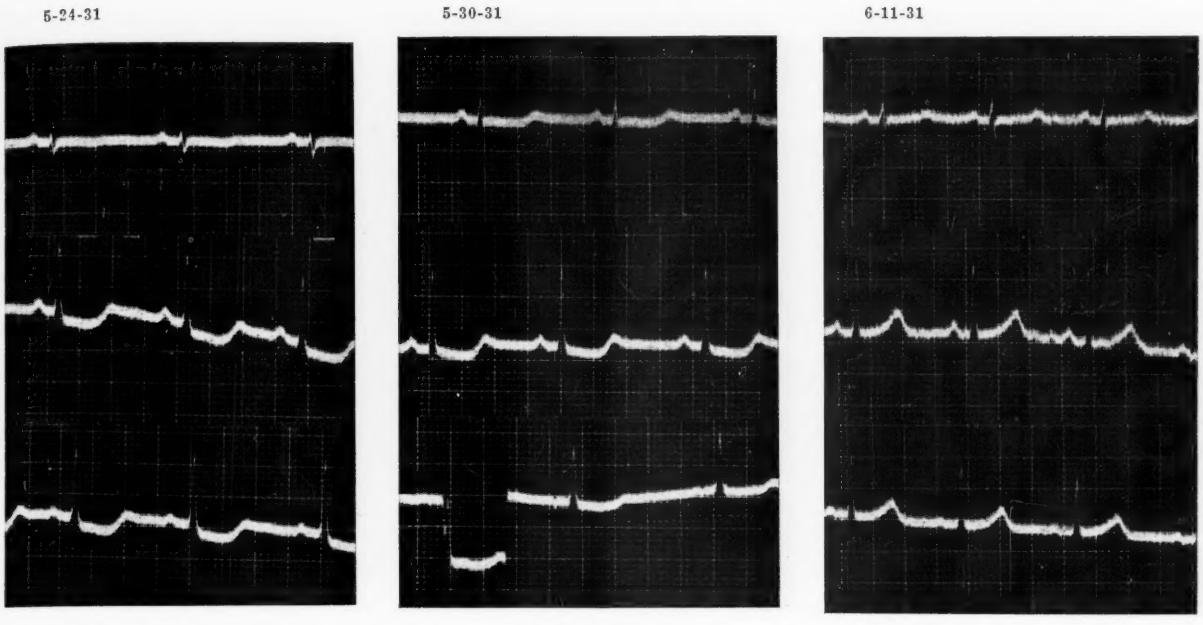
Discharged August 4, 1931.

The electrocardiograms showed a gradual drop in voltage from 1.8 m.v. to 1.1 m.v. and this had not returned to normal in June, 1931. Characteristic changes in the T waves. The PR interval was 0.14 to 0.16 sec. and the Q.R.S. was 0.05 to 0.06 sec. The heart changes were much less than expected and the electrocardiogram finding proportionately less than the clinical evidence. No block occurred and the patient recovered.



M. R.

PLATE VII.



M. R.

PLATE VIII.

CASE 4. Plates IX, X and XI.

T. K., aged 5 years, admitted February 11, 1932 with extensive faucial membrane over tonsils, pillars, uvula, and pharyngeal wall of five days' duration. There was serosanguineous nasal discharge. Thirty thousand antitoxin units given intravenously on admission.

Cardiac onset on ninth day after onset with pallor, apathy, weak first sound and a third heart sound. February 17, 1932 vomiting and more prostration. Showers of rapid beats at times. Palatal paralysis.

Heart area normal.

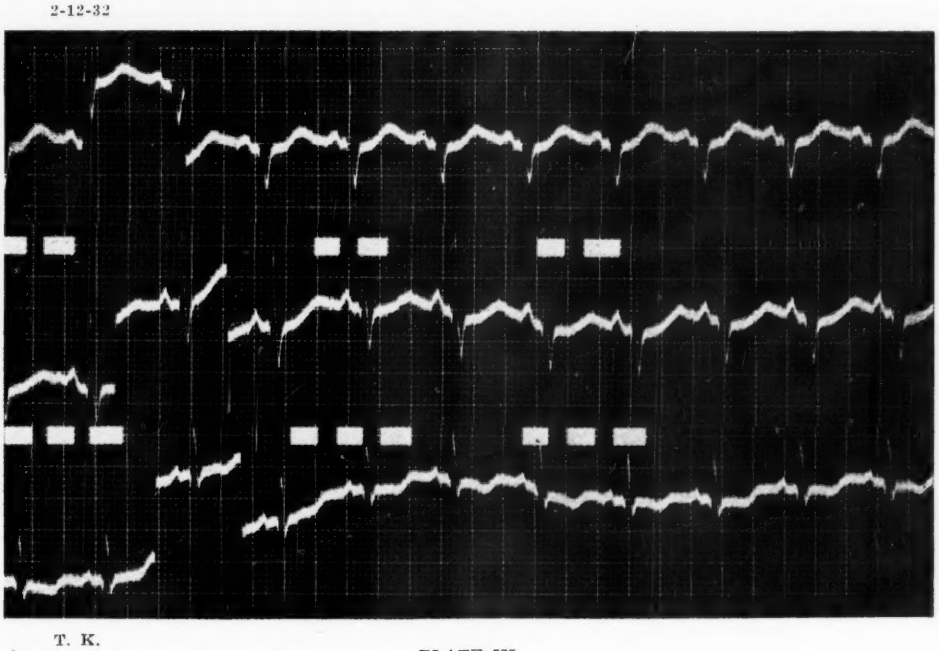
Blood pressure

2-11-32	90-48	2-16-32	47-30
2-13-32	70-37	2-17-32	60-28
2-15-32	60-30	2-18-32	60-42

Died suddenly February 18, 1932 two hours after the last electrocardiogram.

The electrocardiograms presented periods of sino-auricular rhythm and of A.V. dissociation block. Q.R.S. varied from 0.12 to 0.15 and PR from 0.16 to 0.20. T waves went through changes seen in myocarditis as inversion, diphasia and low origin and the voltage dropped to 0.5 m.v.

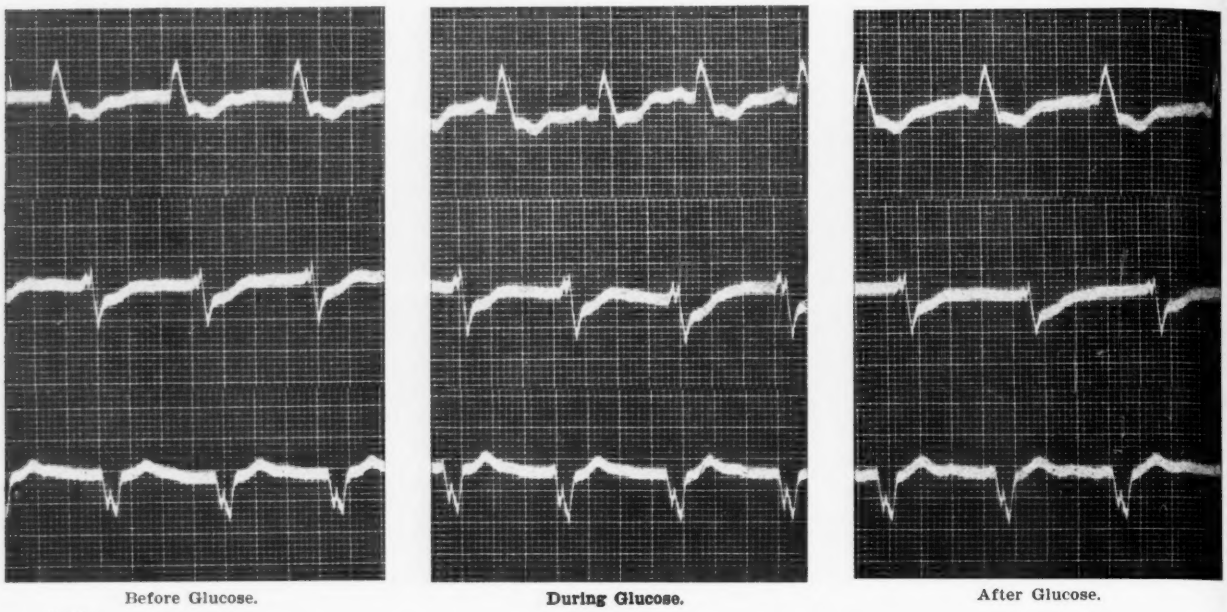
There was marked damage both to the myocardium and the conducting mechanism.



T. K.

PLATE IX.

2-16-32

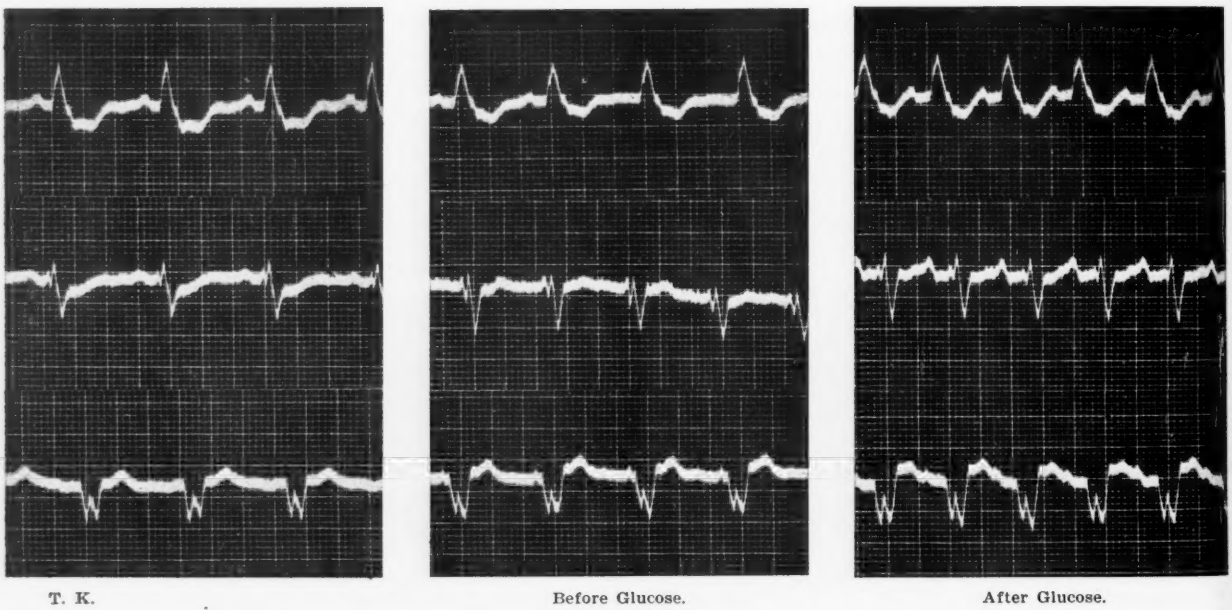


Before Glucose. **During Glucose.** After Glucose.

T. K.

PLATE X.

2-17-32 2-18-32 2-18-32



T. K. Before Glucose. After Glucose.

PLATE XI.

CASE 5. Plate XII.

A. S., 4 years, admitted January 8, 1932 with thin membrane over tonsils, uvula, pharyngeal wall and nostrils of seven days' duration. Forty thousand antitoxin units given intramuscularly on admission. T. normal. Hemorrhages from mouth, lips and into skin. Bleeding time eight minutes. RBC 3,800,000. Platelets absent. Red cells normal. WBC 46,400; polys. 71.5 per cent, Lymph. 25.5 per cent; Monocytes three per cent.

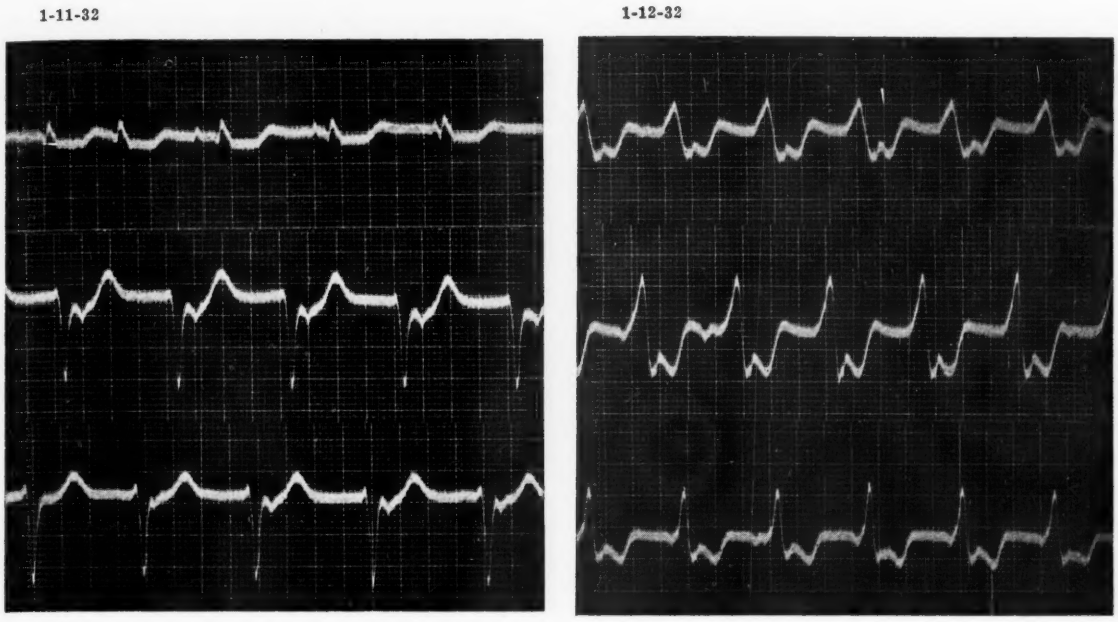
Cardiac onset ten days after onset; apathy, pallor, weak first sound, no enlargement.

Blood pressure

1-11-32	72-48
1-12-32	62-38

Died suddenly January 12, 1932.

Electrocardiograms show AV and intraventricular block. Auricular rate 69 and ventricular rate 77 to 88. Q.R.S. from 0.11 to 0.24 sec. In this case severe conducting tissue damage occurred with A.V. and bundle branch block with very little change in the clinical cardiac signs and with little general evidence of the seriousness of the condition. Up to the time of his death, a bedside observer would not have guessed the situation except through experience with diphtheria heart damage.



A. S.

PLATE XII.

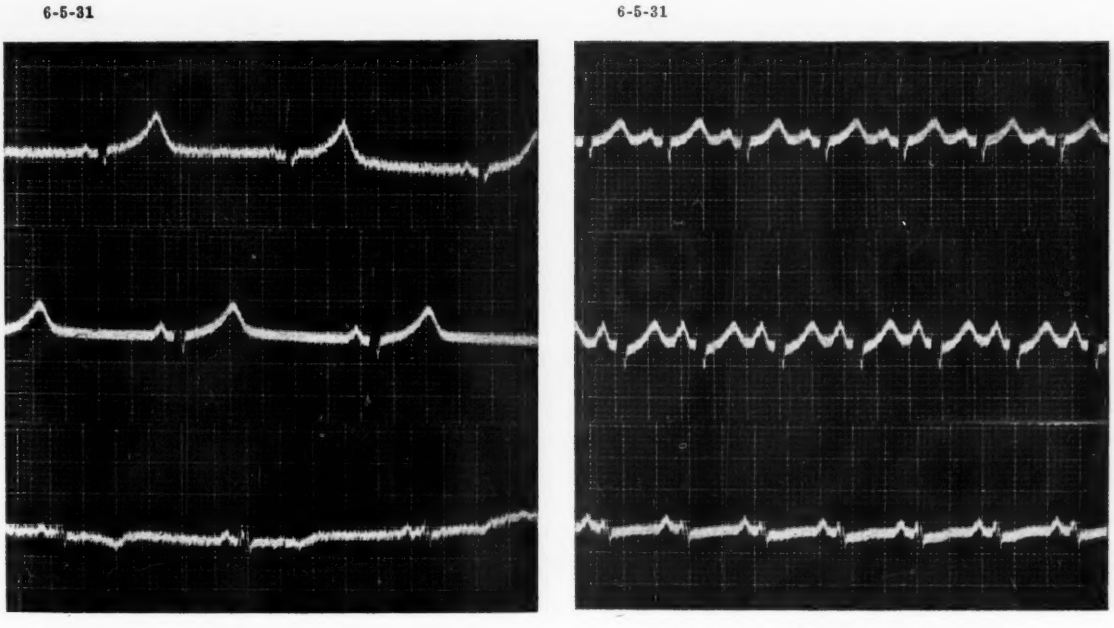
CASE 6. Plate XIII.

J. B., 17, admitted May 31, 1931 with extensive diphtheritic membrane extending over left side of throat and swelling of throat and neck of four days' duration. T. 100°.

The pulse rate dropped from 80 to 50 by the eighth day after onset. No cardiac symptoms were found. The blood pressure dropped from 90-40 to 78-28. The pulse gradually rose to normal in three weeks and the pressure to 100-40.

Palatal paralysis was present at two weeks and persisted for one week.

The electrocardiogram was normal except for the bradycardia. Atropine was given to remove vagus control and it required 1/25 gr. intravenously in two hours. As no vagus paralysis appeared during the convalescence, this was considered simply as a convalescent bradycardia.



Control.

After gr. 1/25 atropine intravenously.

J. B.

PLATE XIII.

CASE 7. Plates XIV, XV and XVI.

M. W., 10, admitted October 23, 1931 with extensive membrane over fauces, palate, and uvula of four days' duration. Sixty thousand antitoxin units given intravenously on admission. T. normal. Throat cleared in five days. Cardiac onset tenth day after onset. Soft first sound, enlargement to left. Vomited November 1, 1931. Pharyngeal paralysis. Third heart sound. Epigastric pain and tenderness over liver.

Gradual improvement in heart sounds and no further circulatory symptoms after the fourth week. Paralysis increased involving face, eyes, and throat with considerable dysphagia. These had disappeared by the end of December.

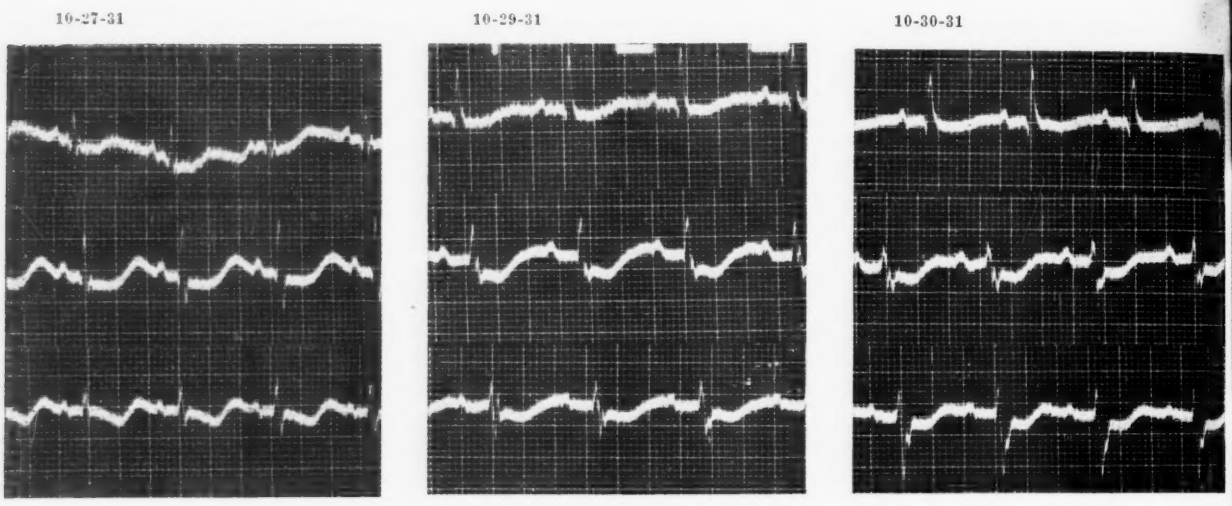
Blood pressure

10-26-31	95-60
10-29-31	85-60
10-31-31	70-50
11- 2-31	67-46
11- 4-31	62-48
11- 7-31	80-50
11-16-31	96-60

Discharged February 15, 1932.

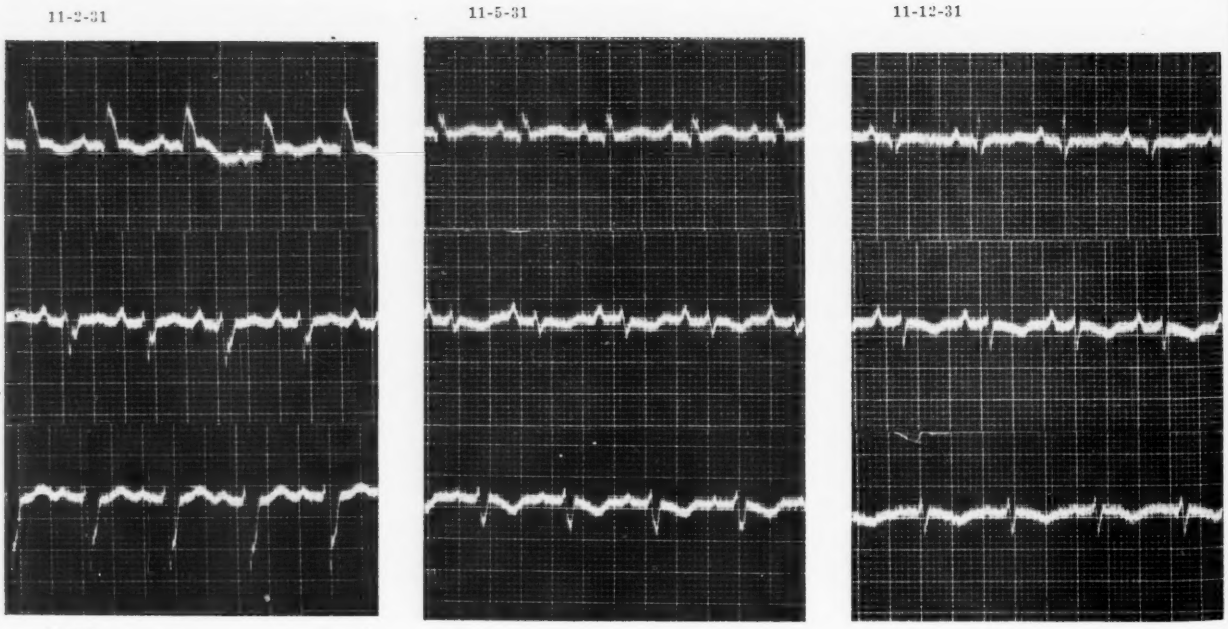
The electrocardiograms showed a slight AV block with a PR of 0.20 second and a Q.R.S. of 0.10 to 0.12 sec., during the first few days only but chiefly the gradual drop in voltage down to 0.4 m.v. and the progressive changes in the T waves with gradual return. The electrocardiograms were essentially normal in two months. There was left axis deviation.

In this case there was a slight transient heart block and the characteristic persistent changes of myocarditis.



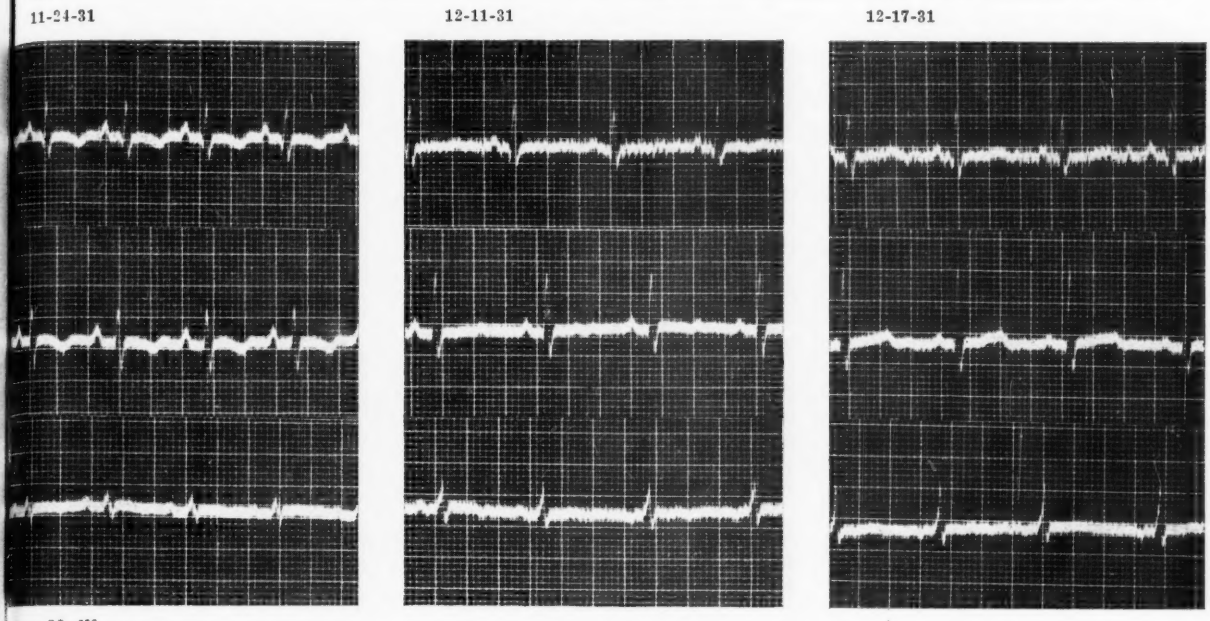
M. W.

PLATE XIV.



M. W.

PLATE XV.



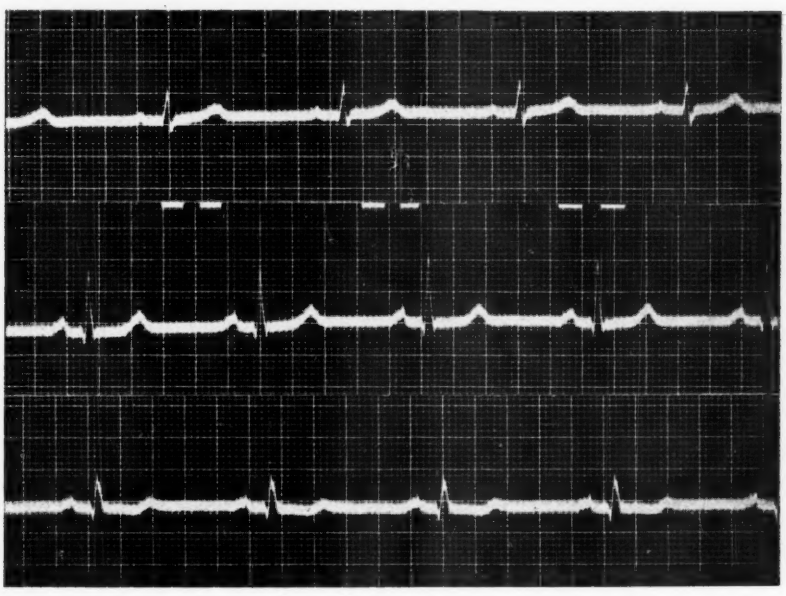
M. W.

PLATE XVI.

CASE 8. Plate XVII.

C. E., admitted October 19, 1911 on the fourth day of diphtheria with extensive membrane over whole throat and great swelling of throat and neck. Four hundred and fifty-eight thousand units antitoxin from the fourth to ninth day after onset. Myocarditis October 23 to November 18. Diphtheritic paralysis very extensive from December 3 to February 4. During the past 20 years since this attack, the patient has had no circulatory defect and is now living an active outdoor life.

The electrocardiogram shows no abnormality.



C. E.

PLATE XVII.

SCARLET FEVER

In scarlet fever, the heart condition is reversed as compared with diphtheria. Myocarditis, alone, is rare. Since electrocardiographic study has been available no well-marked case has occurred in our wards in several thousand cases. Septic endocarditis and pericarditis and subacute bacterial endocarditis, although less rare, are not considered in this account.

The characteristic heart damage in scarlet fever is a benign endocarditis. In recent years this has occurred in less than 0.1 per cent, while in earlier periods from 0.25 to 0.5 per cent.

The diagnosis is based on the usual findings of fever, cardiac murmurs, with tachycardia, changes in heart size and associated arthritis. Difficulties in diagnosis are often great and the determination of a true endocarditis from the so-called functional murmurs uncertain. Murmurs during diastole, replacement of the heart

sounds by the murmur, persistence of murmur through systole, and cardiac enlargement are usually considered indicative of structural change in our cases. Electrocardiographic changes are extremely rare although in some of the cases too few records were taken to exclude such evidence.

The onset is typical during the second and third weeks after onset. Association with arthritis is very common, but the details are not yet complete in the study of our cases. The course is usually favorable and it is rare to find active disease persisting more than a few weeks. A series of cases, now being studied and extending back several years, does not show great frequency of recurrent attacks.

The crippling effect varies as in other forms of endocarditis, but it is slight compared with rheumatic fever.

The chief interest is the relation to rheumatic fever. Until the etiology of rheumatic fever and of the endocarditis of scarlet fever is known, we can only speculate on the relationship. It is obvious that scarlet fever itself is of much less importance in the acute endocarditis of childhood than rheumatic fever, even granting specific causes for each.

The problem is still further complicated by the fact that undoubted rheumatic fever follows scarlet fever at times as well as that through accident or otherwise scarlet fever follows attacks of rheumatic fever.

There are several points which may indicate that the endocarditis of scarlet fever is specific for that disease. It occurs at a fairly characteristic time during the disease, it runs a fairly uniformly short active course, no subcutaneous nodules have yet been found in our series, and the arthritis is self-limited, rarely, if ever, extending over a period of more than from one to three weeks.

THE HEART IN PULMONARY TUBERCULOSIS*

BY JOHN B. HAWES, 2ND, M.D.†

THERE is comparatively little literature on the subject of the heart in tuberculosis when one considers the vast amount that is constantly being done and written concerning all phases of tuberculous disease. I freely admit, after some twenty-five years of intensive dealing with tuberculosis, that it has been only in rare cases that the heart per se demands attention and specific treatment. As is well stated by Thomas Ziskin (The heart and tuberculosis. *Journal-Lancet*, 44:151, 1924) "the problem of the heart in tuberculosis is not so much a problem of the heart but of the disease itself".

Much has been written concerning the small size of the heart in tuberculosis. Some are of the opinion that it is the smallness of the heart, either a congenital or an acquired abnormality, with the consequent poor circulation in the lungs and elsewhere that paves the way for the development of tuberculosis later on. Others are equally certain that the small size of the heart muscle is simply the result of a tuberculous toxemia producing wasting away, loss of tone and deterioration of muscular tissue all over the body including of course the heart muscle. Doubtless, both viewpoints are correct in individual cases although I am strongly of the opinion that in the vast majority of instances the latter conception is the correct one and that the heart becomes small and its muscle weak and flabby as a result of long-continued dosage with the toxins of the disease. It is certainly the continued action of such toxins, causing degenerative changes in the heart muscle, that produce most of the symptoms of cardiac origin met with in the course of tuberculous disease.

Whether there is tachycardia or in rare cases a true bradycardia (I have never seen this latter phenomenon) and whether one is due to the stimulation of the sympathetic and the other to stimulation of the vagus according as one is sympatheticotonic or vagotonic, I consider to be a question of academic interest only and one that is not of any clinical importance.

Therefore, to sum up this discussion so far I would state, first, that there are undoubtedly a few individuals of the so-called "sthenic habitus" who possess a small heart and in consequence a poor circulatory apparatus. Such individuals may come down with tuberculosis. Secondly, in most instances, a small heart in tuberculosis is due to the wasting effect on the heart muscle just as leg, arm and shoulder muscles waste away from the same cause. Thirdly, a rapid heart rate is due to a combination of a weak muscle and a toxic stimulation of the sympathetic nervous system. The small heart in tuberculosis has long interested medical men. Laennec speaks of it and in a delightful volume by James, later Sir James Clark of London, written in 1830, I find the following: "The state of the circulation is subject to great variety. In hereditary cases, I think the powers of the heart are commonly under the ordinary standard, whilst the frequency of the pulse is generally above it, and palpitation is not an unfrequent symptom. A small feeble heart I consider a strong, predisposing cause of consumption."

*Read before the New England Heart Association, Cardiac Course, March 2, 1932.

†Hawes — President, Boston Tuberculosis Association. For record and address of author see "This Week's Issue," page 900.

So much for the small heart and rapid or rarely abnormally slow pulse in tuberculosis.

Now, let us consider that tradition, I use the word advisedly, that chronic valvular heart disease especially mitral stenosis is incompatible with pulmonary tuberculosis. This was of course based on the theory that an increased blood supply to the lungs as in a chronic passive congestion would militate against any invasion by the tubercle bacillus. It was Rokitansky who in 1855 first advanced the theory that tuberculosis of the lung and heart disease did not occur together. For years this theory was accepted. Certainly, when I attended the Harvard Medical School (1903) I was so taught. Enough work has been done of recent years, however, to alter our views on this point. Lawrason Brown out of 7115 autopsies on tuberculous subjects found 0.9% of valvular heart disease; Norris out of 8154 autopsies found 3.5%; he likewise tabulated 449 cases of pulmonary stenosis and found that 33% were associated with tuberculosis. Anders reports six cases of valvular disease, 4 of mitral insufficiency, and 2 of mitral stenosis with pulmonary tuberculosis. He concludes that the law of Rokitansky is untenable and that "the association of phthisis with primary valvular heart disease is probably more common than the usual clinical findings have thus far indicated". This is a sane point of view and represents what I believe to be the truth. One may well bear in mind, therefore, based on what I believe to be very good evidence, that when a man known to have valvular heart disease has a frank hemorrhage, tuberculosis should *not* be at once ruled out as its cause. I regret to say that in Massachusetts tuberculosis is too often not diagnosed under such conditions. Various attempts have been made to prove that frank pulmonary hemorrhages were as common in mitral stenosis as in tuberculosis. They have proved nothing and have only done harm. I will continue to maintain that in probably well over 95% of cases a hemorrhage from the lungs of at least a teaspoonful means tuberculosis of the lungs and nothing else.

The next phase of this problem and by all means the most important is that of a heart handicapped, embarrassed and tied down, and often drawn out of place by adhesions occurring in the course of a chronic fibroid phthisis. Yet it is only in exceptional cases that such a heart produces symptoms requiring treatment. In my experience in pneumothorax work, for instance, the heart may be pushed two or three inches to right or left as the case may be without producing cardiac symptoms of any kind whatsoever. In the x-ray films of almost the majority of patients with a chronic fibrosing process, especially if at an apex, the heart, trachea and mediastinum may be seen drawn to one side or the other and yet the patient goes on his way quite oblivious of the abnormal state of affairs inside his chest and usually quite without undue cardiac symptoms. Occasionally, there is pain, dyspnea and some irregularity apparently due to such a cause which is helped by digitalis. Under such circumstances in the vast majority of cases, the best thing to do is to show the patient his x-ray films, explain to him the exact state of affairs and the reason for his symptoms and let him realize his own limitations and that he must learn to live within them. H. S. Bennett (Heart in tuberculosis. *Southwestern Med.*, 8:57 Feb. 1924) states that "the causative factor in the symptomatology and morphology of the heart in tuberculosis is a complex of the physiological and neurological, containing elements of both the somatic and psychic". With this viewpoint I am in hearty accord.

To be successful in tuberculosis work one must treat, first, the patient, and secondly, the disease. Although this subject of the heart in tuberculosis, whether small as is often the case, or of normal size which is still more common, or enlarged as may occur in advanced disease, is of great interest and furnishes ground for much interesting speculation, I would still maintain that one should treat the patient first, his tuberculosis second, and his heart rarely if ever.

Discussion

Conrad Wesselhoeft, M.D.: What Dr. Place has said has been most interesting and enlightening. The problem of the circulatory failure in diphtheria is far from any satisfactory solution. There are two melodramatic events which occur on diphtheria wards. The first is when a child dying of laryngeal diphtheria is rescued by intubation. The second is when a child who has been rescued by means of antitoxin, and to all appearance has recovered, the membrane having disappeared, suddenly exhibits that curious expression, pain in the epigastrium, perhaps vomiting, gallop rhythm or other disorders of rate and rhythm, and in spite of all efforts dies in a comparatively few hours. Our measures of dealing with the initial stages of diphtheria are in decided contrast to our complete inability to deal with this situation once it has arisen.

From 1913 to 1931 inclusive, we have had 5,952 cases of diphtheria at the Haynes Memorial, with a death rate of 9.7 per cent. Of the total number 11.6 per cent showed disturbance of rhythm, and of these 686 cases which developed disturbance of rhythm 44.3 per cent died. Of course, the death rate of diphtheria is largely influenced by the per cent of laryngeal involvement. Of the 586 diphtheria deaths during this period, 47.4 per cent had laryngeal involvement. The mixture of laryngeal and circulatory factors makes it impossible to present figures of value.

Many measures have been tried with the hope of overcoming circulatory failure. Drugs of the digitalis group have been found unsatisfactory, as have also adrenalin, strychnine, caffein, and alcohol. Barium chloride has been tried as a vasoconstrictor, and nitroglycerine as a vasodilator, with equally bad results. Working on the assumption that the condition was a "toxemic shock," Harding urged raising the foot of the bed. Impressed by this logic we began raising the foot of the bed in cases of circulatory failure in 1923. Instead of improving our death rate we find that during this later period, covering 1906 cases, we had 199 deaths with a death rate of 10.4 per cent. Consequently, the figures do not seem to indicate that we have accomplished anything by this method. It has often seemed questionable whether the theoretical value of raising the foot of the bed was worth considering, when so many children will use up muscular energy trying to get their heads at the raised end of the bed. I feel that this method has proved useless. (Intravenous glucose, so strongly recommended by some, has also proved worthless in my hands.) The case that recovers does so under his own power assisted by rest in bed. Dr. Place has shown that the electrocardiogram can help in the matter of prognosis.

The tragedy of late circulatory failure is overwhelming. It is all very well to say that it could be prevented, for the prevention of diphtheria naturally prevents the cardiac failure which accompanies or follows the disease, but those of us who are called upon to treat advanced cases of diphtheria in contagious hospitals still have to deal with this serious problem.

SMALLPOX STATISTICS

In the May issue of the Statistical Bulletin of the Metropolitan Life Insurance Company appear certain statements concerning smallpox in this country and Canada in 1931 which are pertinent. This Bulletin covering reports from forty-three states, the District of Columbia and eight Canadian provinces, indicates a decided decrease in the prevalence of smallpox during 1931. It is important that lessons of the past should be carefully considered.

In the first place the fatality rate of smallpox has shown no appreciable reduction and, secondly, those states which show the greatest number of deaths from smallpox are those in which there is no general acceptance of vaccination and revaccination. This is especially shown in Indiana where there were 3,000 cases, Iowa and Kansas well over 2,000 cases each and Illinois, Ohio, Oklahoma and Texas very close to 1,000 cases each.

It is significant to compare these states with a group of fifteen states along the Atlantic seaboard where the population is approximately the same as that of another group of nineteen, that is, 48,900,000 against 48,711,000. It is on record that only 1,063 smallpox cases were reported in these Atlantic states as against 22,413 in the central and western group. In other words the case-incidence rate in these eastern seaboard states is only two per 100,000 as against 46 per 100,000 in the central and western group. So far as the three Pacific Coast states are concerned in comparison with the Atlantic seaboard, it is found that there were reported more than three times as many cases of smallpox in the former area as in the latter, although the population of the Pacific states is only one-sixth that of those on the Atlantic seaboard.

In the state of Washington alone there were 1,112 cases where the population is but 1,589,000. If these statistics should be carefully studied and their application made it would be more generally accepted that vaccination is the great preventive against smallpox as has been so frequently brought out by Dr. Samuel B. Woodward of Worcester.

The public should be led to realize that the superior quality of vaccine now available entails no great inconvenience and the application requires only a very few minutes. While we hope that this protective measure will in the future be universally adopted, we have to recognize the impedimenta exhibited by our opponents.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending October 29, indicate a mortality rate of 10.3 as against a rate of 10.9 for the corresponding week of last year. The highest rate (21.3) appears for Nashville, Tenn., and the lowest (5.9) for Detroit, Mich. The highest infant mortality rate (10.7) appears for Atlanta, Ga., and the lowest for Bridgeport, Conn., Duluth, Minn., Fall River, Mass., Long Beach, Calif., Miami, Fla., New Bedford, Mass., Paterson, N. J., Springfield, Mass., Tampa, Fla., and Yonkers, N. Y., which reported no infant mortality.

The annual rate for 85 cities is 11.1 for the forty-three weeks of 1932, as against a rate of 11.9 for the corresponding period of the previous year.—*Bulletin Bureau of the Census.*

CONSERVATIVE KIDNEY SURGERY*

BY AUGUSTUS RILEY, M.D.†

CONSERVATIVE kidney surgery, particular resection for large solitary kidney cysts, heminephrectomy for double kidneys and ureters, if one part of such double kidney has become pathological, and plastic operation on the kidney pelvis, is demanding a great deal of attention. Careful studies of each individual case, improved diagnostic aids and recent research in kidney work have been important factors in bringing about this conservatism. One must be able to interpret all of the existing pathology so as to be conservative, yet be able to remove the existing diseased portion of the kidney.

In every large urological clinic one sees many kidney abnormalities. These abnormalities often encountered are duplication of calices, double kidney with a single pelvis, double kidney with double pelves and a single ureter, double kidney with double ureters uniting in a common ureter some distance from or close to the bladder, double kidneys and ureters with both ureters entering the same side of bladder or different sides of the bladder, and horseshoe kidneys. Some portions of these abnormalities often become pathological and call for surgical interference to relieve or eradicate the diseased part and put the individual in better physical condition than before such surgical interference was undertaken.

There have been several cases reported which were operated on; attempts to remove these pathological parts and the results have been most encouraging to future attempts at this type of surgery.

Young[1], in 1917, reported a case which he operated on. He removed the upper portion of a double kidney for a large branching stone. He says, "During recent years we have had in our clinic several cases of double kidneys in which one portion of the double kidney was diseased. Only one case came to operation. A careful study of the literature shows that although the condition is not so rare the recognition and cure by operation is extremely rare." He claims his case to be the only one reported in full up to that time.

Walters[2], in 1930, reported three cases in which resection of the diseased portion of a duplicated kidney was done; in one case the kidney was resected for a large cyst of the lower pole. He says, "Ideal cases for heminephrectomy would seem to be those in which a complete duplication of the renal pelvis and ureter is present and in which it is necessary to remove only the portion of the kidney involved. Large solitary cysts and that portion of the kidney containing the cyst should be resected whenever possible instead of removing the whole kidney."

He made preoperative and postoperative studies on the functions of his cases and found that resection did not interfere with the function of the remaining portion.

Judd[3] says, "Resection of a double kidney is practical, providing the diseased portion is definitely confined to one segment. It is entirely feasible to perform a resection in cases of solitary kidney and it should be done whenever possible." He reports the resection of the remaining kidney in a patient who had a normal blood urea before operation, and found a distinct change in the blood urea after operation. The changes were as follows: second postoperative day Mg. per. 100 cc., fourth postoperative day 92 Mg. per. 100 cc., ninth postoperative day 96 Mg. per. 100 cc., and thirteenth postoperative day 44 Mg. per 100 cc., showing that renal function was definitely interfered with by operation but by the thirteenth postoperative day there was practically a return to normal. He claims that a more complete recovery of the function can be expected.

Quinby reports cases of plastic operation between kidney pelvis and ureter for obstruction at the uretero-pelvic junction with good results. He refutes Hinman's theory that a kidney thrown out of commission does not regain its function because the hypertrophy of the other kidney has taken up the necessary work.

Young[4] at a recent date reported two cases, one a double hydronephrosis due to obstruction by an aberrant vessel at the ureteropelvic junction, the other a unilateral hydronephrosis due to an aberrant vessel at the ureteropelvic junction. The function of the right kidney of the double hydronephrosis was practically nil when first examined. With ureteral catheter drainage of this kidney for a long time the function reappeared and improved so much that Young did a plastic on the kidney pelvis, reducing the pelvis in such a way as to draw the ureter away from its obstructing aberrant vessel. From such startling results, Young advocates a plastic on the kidney pelvis only and not a ureteropelvic plastic as advocated by Quinby[5] and others.

We are much indebted to Herbst and Polkey[6] for their experimental work on kidneys to determine the possibility of kidney resection and the results of such resection on the relative function of the remaining portion of the kidney and the results on the hypertrophy of the kidney not operated and the atrophy of the kidney op-

*Read before the Clinical Staff Meeting, Boston City Hospital, February 18, 1932.

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erated. For their experiments they used thirty-seven dogs. Two of the dogs died under ether. One died of postoperative hemorrhage, and one died of peritonitis, leaving thirty-three dogs from which they were able to draw their conclusions.

Their conclusions were as follows: There was no complete atrophy or complete loss of function of the resected kidneys. There was no

FIG. I. Pyelogram of right kidney showing three normal appearing calices in the lower one-half of kidney and abscessed calices in the upper one-half of kidney.

compensatory hypertrophy of the other kidney. Resection reduces the weight and function of the kidney. The resected kidney showed a diminished function, which was most marked during the first two weeks. The diminished function was due to congestion and repair process. Mattress sutures caused most kidney destruction. Stripping the capsule and making a V-shaped incision seem to give the best results. There was never a urinary fistula from healthy kidney tissue.

From these findings Herbst believes that resection of a sacculated portion of a kidney for stones is better than pyelotomy.

I have been able to find three cases which I felt were proper cases for resection, two for sacculated stones in the calices; one with a large stone in the upper calix, the other with a large stone in the lower calix. One of these two cases I have been able to examine some months after operation and the results have been most gratifying. The second of these cases was done only recently and it is too soon to know what the final results will be.

In the third one of these cases, after a very careful study, I was able to do a resection of the right kidney, removing more than the upper one-third of the kidney. The kidney was one of the abnormal types that we occasionally see, part of which had become pathological. Pyelographic studies showed six major calices in each kidney instead of three major calices found normally in each kidney. There appeared two definite sets of three calices each in each kidney. Each set of calices appeared to have its own pelvis, joining to form a common pelvis for the whole kidney. The three calices forming the upper part of the right kidney showed a destructive process consistent with tuberculosis of the kidney or simple abscess formation (figures I and II). The three calices forming the lower part of the right kidney appeared normal, there were good cupping and no irregu-

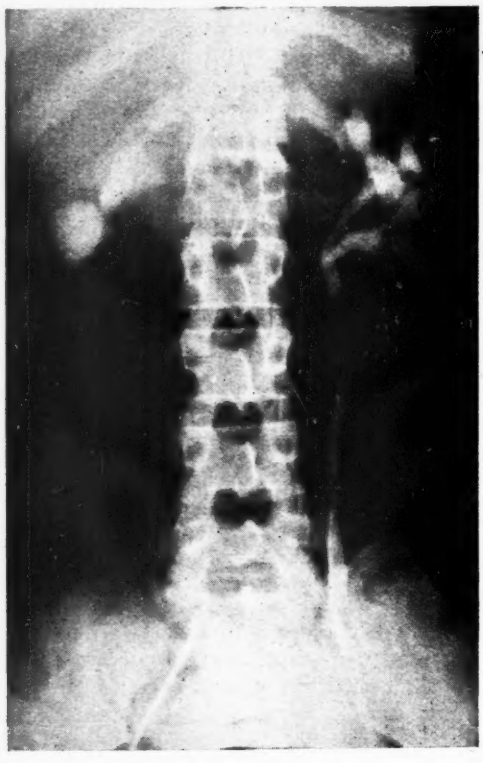

FIG. II. Shows double kidney (left) with six normal appearing calices, and retained abscess of upper calices of right kidney.

larities in their outline. The kidney pelvis appeared normal.

At the time the first pyelograms were made both ureters were catheterized with No. 7 catheters, which were passed easily without meeting any obstruction. Urine from the left kidney was clear. Urine from the right kidney was clear at first. Drainage from the right kidney after the sodium iodide had been injected was turbid, which no doubt meant that

distending the kidney pelvis with sodium iodide for the pyelograms caused the abscess to drain.

Microscopic examination of the urine showed a rare pus cell from the left kidney, and an occasional pus cell from the right kidney in the specimen obtained at the first drainage. The specimen obtained from the right kidney after injecting the sodium iodide was turbid and loaded with pus.

Five days after the pyelograms were made, the patient was cystoscoped again. Both ureters were catheterized and specimens of urine were obtained for guinea pig inoculation, and a split function was done. The right kidney showed 20 per cent function, the left kidney showed 30 per cent function in the first fifteen minutes. The appearance time of each kidney was three minutes.

The essential points in the history of this case are as follows:

A colored girl, nineteen years of age, was admitted to the Fifth Surgical Service, Boston City Hospital, September 17, 1931. Her chief complaint was pain in the right side of abdomen. The first attack of pain occurred about four months ago. The pain at that time was constant for a day and has recurred at different times since. The night before admission she had a severe pain in the right upper quadrant of the abdomen. She was unable to sleep because of severe pain. There has been some nausea but no vomiting during the past few days, and burning urination but no frequency for the past two days.

Physical examination was negative except for the abdomen. There were some tenderness and spasm in the right costovertebral angle and lower abdomen. No mass was felt. Temperature 98.5°. Pulse 88. Respiration 22. White blood count 9600. Urine showed a rare white blood cell.

The patient remained in the hospital until October 3, 1931. At no time did she show any rise in temperature or increase in pulse. She was discharged to the Out-Patient Department for observation until a guinea-pig report could be obtained.

She was readmitted November 10, 1931, to the Fifth Surgical Service.

She had had some pain in her right side and back constantly since leaving the hospital. Burning and frequency of urination have increased. Nocturia is now three to four times.

Physical examination at this time was practically the same as at the first admission. The urine from the bladder was now loaded with pus, N.P.N. 20.

November 21, 1931. Operation. Anesthesia: Spinal Novocaine. Hockey-stick incision was made to expose the right kidney. The kidney was easily exposed. On the convex surface of the kidney was a thin-walled area about the size of a fifty-cent piece. Palpation of the kidney pedicle revealed two large pulsating arteries, one apparently leading to the upper pole, the other leading to the lower pole of the kidney. The artery thought to supply the upper pole was not disturbed, as we did not know just how much of the kidney was supplied by each artery. The thin-walled area was opened and some pus was evacuated. Exploring the abscess cavity three distinct compartments could be made out, corresponding to the pyelographic findings (figures I and II).

The upper portion of the kidney was resected, removing the abscess cavities which took up more than the upper third of the kidney, down to normal appearing kidney tissue, showing plainly the markings of normal kidney tissue.

Hemorrhage was easily controlled by well-placed through and through No. 2 chromic catgut sutures. There was one profuse bleeding point which corresponded, I thought, to the upper artery felt in the pedicle. This was easily controlled with a suture ligature.

The cut edges of the kidney were easily approximated with No. 1 chromic catgut interrupted sutures. The kidney was held high up in place

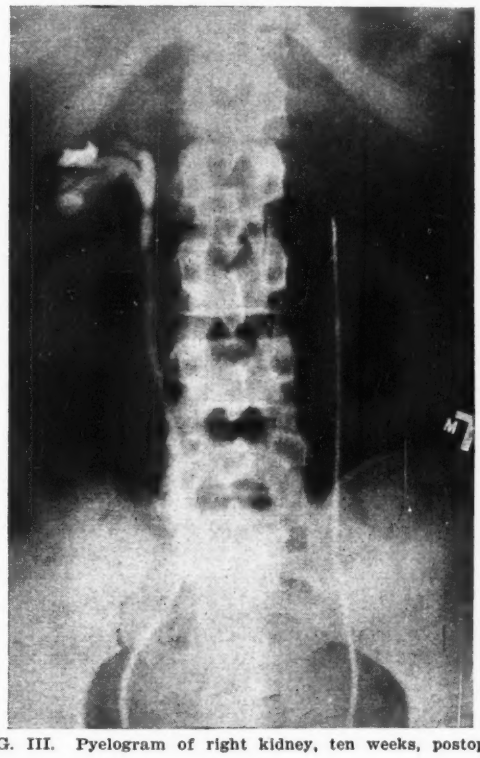

FIG. III. Pyelogram of right kidney, ten weeks, postoperative.

(nephropexy), by suturing the fatty capsule under it. A cigarette drain was placed near the kidney. The wound was closed by approximating the muscle edge with No. 2 chromic catgut interrupted sutures. The skin edges were approximated with interrupted silkworm gut sutures.

After operation the patient showed a rise in temperature for the first six days. The cigarette drain was removed on the sixth day. There was a great deal of serosanguineous fluid following the removal of the drain. There was an immediate drop of her temperature to normal. There was never any odor of urine on the dressing. The wound healed without any suppuration. The patient was discharged from the hospital fifteen days after operation.

After leaving the hospital the patient was seen by me several times in the Out-Patient Department. She has been free of pain and all urinary disturbances.

January 28, 1932, about ten weeks after operation, cystoscopic examination was done and kidney studies were made. Both ureters were catheterized with No. 7 catheters. Specimens of urine were obtained from each kidney. Urines were clear with no shreds. Centrifuged sediments showed a rare white blood cell in each specimen. Phthalein test showed 35% from the right kidney, 50% from the left kidney. N.P.N. 26.

Pyelograms of the remaining portion of the right kidney (figure III) show a normal number of calices and a normal-sized pelvis with the remains of the draining channel of the upper calices which were removed.

CONCLUSIONS: The opportunities for conservative kidney surgery are not very common, but from the remarkable clinical results obtained in the cases operated upon and reported, such surgery should be undertaken, provided that after careful studies all diseased parts can be fully removed.

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TREATMENT OF ACUTE ANTERIOR POLIOMYELITIS WITH NON-SPECIFIC PROTEIN. A PRELIMINARY REPORT*

BY C. L. THENEBE, M.D.,† AND V. P. CENCI, M.D.†

DURING the latter six months of the year 1931, Connecticut was the seat of a poliomyelitis epidemic. This epidemic was of mild intensity based upon the fact that the number of cases showing paralysis was low.

Dr. Millard W. Knowlton, Director of the Bureau of Preventable Diseases, Connecticut Department of Health, has furnished the following statistics:

TABLE 1

CASES OF POLIOMYELITIS REPORTED IN CONNECTICUT—1931

Month	Cases
January	0
February	0
March	0
April	1
May	1
June	2
July	49
August	421
September	463
October	153
November	35
December	8
Total	1133

HARTFORD ISOLATION HOSPITAL REPORT

Two hundred and seventy patients were admitted to the Hospital during the year 1931; seven of these during July; 119 during August; 96 during September; 37 during October; eight during November and the remaining three patients during December.

There were 94 preparalytic patients (23.8%). There were 176 with demonstrable weakness or paralysis (65.2%).

There was a total of 16 deaths (5.91%). One death occurred in the preparalytic group (0.37%). The remaining 15 deaths occurred in the spinal, bulbar and cerebral forms of the paralyzed group (5.5%).

The following report includes the group of preparalytic cases treated with sterile milk administered intramuscularly. One of us (Thenebe) believed that non-specific protein therapy could be tried to accelerate immune body formation. This group represented almost wholly consecutive cases. Consequently this report is not so valuable as it would have been if control cases had been included.

SUMMARY: A total of 33 cases treated with sterile milk intramuscular injections has been reported. Appreciable paralysis or weakness was noted in 6% of these cases during hospitalization. At a later examination‡ slight weakness was noted in an additional 18% of these cases. There was no mortality. The spinal fluid cell count varied from 10 to 310 cells. The cases in this group presented less weakness or paralysis than the other groups (serum and control)[1] but the number of cases reported was not sufficient to draw any definite conclusions.

The symptoms and physical signs were present, as accurately as could be determined, in the following percentages. Admission temperature present in 87%; diarrhea in 2%; constipation in 2%; drowsiness in 13%; headache in 70%; nausea in 53%; vomiting in 45%; pallor-circumoral in 93%; prostration in 13%; restlessness, irritability and anxiety in 52%; stiff neck in 93%; stiff back in 83%; sore throat as a complaint in 37%; hyperemia and edema of pharynx in 93%; tache cérébrale in 93%.

GENERAL COMMENT: The milk treatment as set forth in Table 3 was used before Kramer[1] had reported his series in connection with which he advocated further study on a larger scale of preparalytic cases treated with human convalescent serum. It is certainly true that serum therapy must be thoroughly studied before de-

*A preliminary report was presented to the Hartford Medical Society September 19, 1931. The muscle examinations during hospitalization of the patient were done by Drs. Thomas P. Moylan and Dr. Maurice F. O'Connell, orthopedists, Hartford. The essential work done by these men was greatly appreciated.

†Thenebe—Visiting Epidemiologist, Hartford Isolation Hospital. Cenci—Resident Physician, Hartford Isolation Hospital. For records and addresses of authors see "This Week's Issue," page 900.

‡The later muscle examinations, October 21, 1931, were done by Miss Kari Hagen, Harvard Infantile Paralysis Commission, to whom we are also truly grateful.

termining the value of any other therapeutic measure, i.e., intramuscular milk injections. However, there will be certain definite problems after a more complete study of the use of any therapeutic measure. It would seem that additional information pertaining to the prophylaxis, early diagnosis and treatment of poliomyelitis is necessary. Draper[2] has recently stated that the diagnosis of poliomyelitis should be made during the preataxic tremor stage of the disease. This is usually an extremely difficult diagnosis to make at this time. Further methods of diagnostic precision often seem necessary to make such a diagnosis. The administration of serum (H.C.S.) when the ataxic tremor stage has been reached would seem unnecessary if the anterior horn cell virus union has already been consummated. However free virus could be neutralized in the bloodstream which might prevent further union of additional anterior horn cells with virus.

The question of intraspinal administration of human convalescent serum arises constantly. Fairbrother and Hurst[3] have shown repeated failures to detect virus in the cerebrospinal fluid. They suggest that the meninges do not play an important rôle in the pathogenesis of poliomyelitis. (This might also be suggested in the low spinal fluid cell count.) They have shown that virus spreads by the nerve fibres from above downward. This might again suggest that virus is carried wholly to the involved region or regions by the blood or lymph stream. If virus is only very rarely demonstrated in the cerebrospinal fluid, does it seem reasonable to administer serum intraspinally? If virus is carried by the blood or lymph stream directly to the gray matter of the spinal cord or brain why could that not be one reason that the intravenous or intramuscular administration of serum should be the logical method of approach? Human convalescent serum administered intraspinally produces a severe aseptic meningitis. This serum is contraindicated in the treatment of bulbar cases because it may increase the round cell infiltration, edema, hemorrhage, etc., at the site of involvement. Could not one argue that the same change may occur when there is spinal involvement?

The future problem of poliomyelitis probably should be directed toward vaccination against this disease unless further information can be

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TABLE 2

AGE DISTRIBUTION OF CASES OF POLIOMYELITIS IN CONNECTICUT—1931

Age in Years		Under 1	1	2	3	4	5	6	7	8	9	10-14	15-19	20-24	25-29	30-34	35-39	40-49	50-59	60+	Age Not Given	Total
State of Connecticut																						
	M	17	8	72	51	50	59	73	39	42	27	98	48	14	8	3	3	8	—	—		620
	F	4	5	61	61	60	50	48	25	39	23	77	20	12	1	5	1	2	—	—		494
	Total																				19	1133
Hartford																						
	M	3	1	5	7	14	12	14	6	6	2	14	5	2	3	—	—	1	—	—		95
	F	—	1	7	10	4	9	12	3	7	4	7	1	6	—	2	—	3	—	—		76
	Total																					171
New Haven																						
	M	2	—	12	10	5	8	11	6	2	4	10	7	2	—	—	—	—	—	—		79
	F	—	—	17	10	11	6	4	2	3	1	9	1	1	—	3	—	—	—	—		68
	Total																				1	148
Waterbury																						
	M	2	1	13	6	5	8	10	2	8	2	12	6	1	—	—	1	—	—	—		77
	F	1	—	6	6	5	3	8	4	3	3	8	1	—	—	—	—	—	—	—		48
	Total																					125
Bridgeport																						
	M	—	1	4	4	6	5	6	1	3	2	3	4	—	—	—	—	—	—	—		39
	F	1	1	6	4	1	3	1	1	1	—	3	2	—	1	—	—	—	—	—		25
	Total																					64
Stamford																						
	M	—	—	3	2	—	1	3	—	—	—	4	1	—	—	—	—	—	—	—		14
	F	—	—	3	1	4	1	—	—	4	1	2	2	—	—	—	—	—	—	—		18
	Total																					32

TABLE 3

MILK CASES

Name	Age	Sex	Color	Cell Count	Globulin	Date of Onset	Date of Admission	Date Treatment Started	Treatment	Date, Paralysis First Noticed; Extent
R. T.	5	M	W	35	+	8-8-31	8-10-31	8-10-31	2 injections — successive days. 3 c.c. each injection.	None.
W. S.	9	M	W	30	+	8-22-31	8-24-31	8-24-31	1st injection 4 c.c., followed by 5 c.c. within 24 hours.	None.
R. E.	3	M	W	10	+	8-12-31	8-13-31	8-13-31	2 injections — 3 c.c. successive days.	None noted until 10-21-31 (3)—Slight weakness lateral abdominal muscles.
E. S.	5	F	C	10	+	8-19-31	8-19-31	8-19-31	3 injections—3 c.c. each on successive days.	None.
L. G.	11	M	W	13	+	8-5-31	8-7-31	8-7-31	1 injection—3 c.c.	None.
R. P.	21	M	W	50	+	8-26-31	8-27-31	8-27-31	1st injection 5 c.c., followed by 6 c.c. within 24 hours.	None.
J. C.	6	M	W	13	+	8-9-31	8-9-31	8-9-31	1 injection—3 c.c.	None.
G. D.	27	M	W	170	+	8-16-31	8-20-31	8-20-31	1st injection 5 c.c., followed by 6 c.c. within 24 hours.	Weakness right and left quadriceps 9-1-31.
R. M.	6	M	W	51	+	8-1-31	8-8-31	8-8-31	3 injections—2, 3, 4 c.c., at 24-hour intervals.	Dromedary type. No paralysis.
A. M.	21	F	W	45	+	8-21-31	8-22-31	8-22-31	2 injections—4 and 5 c.c., at 24-hour intervals.	None noted until 10-21-31 (3)—Slight weakness both hip flexors and abdominals.
G. D.	4	F	W	146	+	8-12-31	8-13-31	8-14-31	2 injections—2 and 3 c.c., at 24-hour intervals.	None.
H. P.	7	M	W	12	+	8-15-31	8-15-31	8-15-31	2 injections—3 and 4 c.c., at 24-hour intervals.	None.
E. W.	12	M	W	21	+	8-30-31	8-31-31	8-31-31	2 injections—3 and 4 c.c., at 24-hour intervals.	None.
E. M.	7	F	W	99	+	8-4-31	8-7-31	8-7-31	2 injections—3 and 4 c.c., at 24-hour intervals.	None.
E. S.	7	F	W	10	+	8-27-31	8-27-31	8-27-31	1 injection—3 c.c.	None.
F. D.	1	M	W	30	+	7-30-31	7-31-31	7-31-31	3 injections—2, 3 and 3 c.c., at 24-hour intervals.	None.
S. H.	3	F	W	50	+	8-27-31	8-27-31	8-28-31	2 injections—2 and 3 c.c., at 24-hour intervals.	None.

TABLE 3 (*Concluded*)

Name	Age	Sex	Color	Cell Count	Globulin	Date of Onset	Date of Admission	Date Treatment Started	Treatment	Date, Paralysis First Noticed; Extent
W. F.	2½	M	W	171	+	8-16-31	8-16-31	8-16-31	2 injections—2 c.c. each, on successive days.	None.
J. P.	5	F	W	60	+	8-31-31	9-1-31	9-1-31	2 injections—2 and 3 c.c. each, at 24-hour intervals.	None.
D. F.	7	F	W	165	+	8-14-31	8-16-31	8-16-31	2 injections—2 c.c. each, on successive days.	8-17-31—Left quadriceps. 8-20-31—Right quadriceps.
R. R.	4	M	W	21	+	9-1-31	9-1-31	9-1-31	1st injection 2 c.c.—2nd injection 3 c.c., at 24-hour intervals.	None noted until 10-21-31 (3)—Weakness both anterior abdominal muscle groups (fair).
R. C.	20	F	W	125	+	8-10-31	8-17-31	8-17-31	1st injection 5 c.c., followed by 6 c.c. within 24 hours.	Dromedary type. No paralysis.
R. O.	5	M	W	217	+	8-16-31	8-17-31	8-18-31	2 and 3 c.c. injected within 24 hours.	None.
H. B.	11	F	W	50	+	8-4-31	8-7-31	8-7-31	3 and 4 c.c. injected within 24 hours.	None.
G. S.	20	M	W	35	+	8-23-31	8-26-31	8-26-31	5 and 6 c.c. injected within 24 hours.	None noted until 10-21-31 (3)—At that time slight weakness right quadriceps was noted.
R. R.	6	M	W	91	+	8-8-31	8-9-31	8-9-31	2 and 3 c.c. injected within 24 hours.	None.
N. C.	13 mos.	F	W	25	+	8-16-31	8-19-31	8-19-31	2 and 3 c.c. injected within 24 hours.	None noted during hospitalization. 10-21-31 (3) slight weakness right anterior and posterior tibial group.
J. C.	4	M	W	50	+	8-29-31	8-31-31	8-31-31	2 and 3 c.c. injected within 24 hours.	None.
L. T.	19 mos.	F	W	70	+	8-11-31	8-18-31	8-18-31	1 injection—2 c.c.	Dromedary type. No paralysis.
S. L.	12	M	W	12	+	8-9-31	8-14-31	8-14-31	3 and 4 c.c. injected within 24 hours.	None.
W. W.	6	M	W	73	+	8-16-31	8-16-31	8-16-31	2 and 3 c.c. injected within 24 hours.	None.
G. S.	6	M	W	315	+	8-9-31	8-12-31	8-12-31	2 c.c. twice at 24-hour interval.	None.
R. S.	15	M	W	225	+	8-23-31	8-24-31	8-24-31	5 and 6 c.c. injected within 24 hours.	None noted during hospitalization. On 10-21-31 (3) slight weakness noted right hip flexors and adductors—Right quadriceps: Right anterior and posterior tibial muscle groups 1.

gleaned in recognizing the disease before the union of the anterior horn cells and virus. Speculatively, when the dosage of virus has been controlled may it not take the form of graduated doses of virus, starting with a minute dose, the virus to be introduced possibly by the inunction route?

SUMMARY: a. A résumé of the poliomyelitis situation in Connecticut and more particularly at the Hartford Isolation Hospital has been reported.

b. A résumé of the cases treated with nonspecific protein (milk injections) has also been reported.

c. Attention should be given to Dr. S. D. Kramer's plea that convalescent serum be used on a larger scale to determine the merits of this type of therapy. Other therapeutic measures can then be studied if this product does not prove to be satisfactory.

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THE SANATORIUM CHILD*

BY LEON A. ALLEY, M.D.†

IN considering the children who have come under our observation at Lakeville, I shall limit this paper to the group under ten years of age who were admitted during the past five years. While we admit patients of all ages, these children in the first decade of life offer a large amount of interesting information. The practical conclusions are the ones I shall attempt to bring out rather than those of any other nature.

During the past five years 178 children have been admitted to Lakeville who were in their first decade of life. The diagnosis of tuberculosis had been made in every case. The average duration of the disease prior to admission was as follows: 34 from one to six months inclusive, 22 from seven to 11 months inclusive, 58 for one year, 30 for two years, the remainder from two to seven years inclusive. One hundred and sixty-nine of these children were white and nine were of the Negro race.

One hundred and thirty-nine of these children had upon admission one lesion. Thirty-four had two or more lesions. Nineteen of these cases were eventually proved to be non-tuberculous. The remaining one hundred and fifty-nine presented tuberculosis in its many and varied forms; some had involvement of the lymphatics and other soft tissues, but in the majority of cases involvement was in the bony structures of the body.

These patients were classified on admission as suffering from tuberculosis of the cervical glands, mesenteric glands, spine, hip, knee, small bones of hands and wrists, small bones of feet and ankles, kidney, and sacro-iliac bones. The fact that but one child in this series had renal tuberculosis is particularly interesting and raises the question as to whether renal tuberculosis is a primary condition or whether the kidney depends for its infection on metastasis from lesions in other parts of the body. Eleven of these children died in the sanatorium. The causes of death were as follows:

Tb. Hip, Amyloid disease, Tb. Meningitis	1
" " Broncho-pneumonia, Tb. Meningitis	1
" Peritonitis	1
" " , Tb. Meningitis	2
" Spine	3
" " , Tb. Knee, Amyloid disease of the liver, Chronic Myocarditis	1
" " , Tb. Ulcers regions of heads of femurs and knees	1
" Sacro-iliac, Tb. Peritonitis, Amyloid disease, Tb. Meningitis	1

The one case of sacro-iliac disease died after a residence of three years and nine months and only adds to the demonstration of the fact that sacro-iliac disease, especially with sinus formation and a mixed infection in this group as in the older groups, presents a very grave prognosis.

Now what other points of interest were noted upon the admission of these children?—First let us consider the source of infection. The original source of infection is either human sputum or cow's milk. Sixty-eight gave a history of other cases of tuberculosis in the family. When we know that the most important etiologic factor is contact with tuberculous adults. must we not assume in this number that the close contact with these cases of human tuberculosis is the answer to the source? But what of the other 110 with no known contact? How many of these cases had unknown contact and how many is it fair to assume were infected from sources other than human beings? Our first thought is of course the milk supply. Did the child receive raw milk or was the milk pasteurized? Primary infection can and undoubtedly does occur in any one of several ways. Cow's milk and contaminated food may be ingested and thus initiate infection by way of the upper and lower digestive tract. This happens often and is a particularly frequent

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source of infection in infancy and early childhood. At every age many may breathe in bacilliferous dust or droplets and thus by the mouth and nose take in bacilli that produce lesions in the upper and lower respiratory tract or in the upper digestive tract. Infants and small children are particularly prone to this type of infection, especially if they are in so-called "tuberculous" surroundings. By contaminating the hands with sputum and introducing this into the mouth, ideal conditions are established for infection. The curve and character of childhood infection bear out the presumption that this method of infection is a common one. Inhalation, ingestion, contact with tuberculous patients and contact with the sputum of the latter all play their part. None of these methods can or should be ignored. These opportunities for receiving tubercle bacilli extend over long periods of years for everyone who is in a "tuberculous" environment. We feel with certainty that congenital tuberculosis plays practically no rôle, but rather that the disease is acquired after birth. Studies[1] indicate that tuberculous infection in infancy is usually a matter of continual exposure, and the number of individuals in any community infected with tuberculosis depends on the opportunities for becoming infected rather than on race or economic status.

Bovine tuberculosis in man is usually of the non-pulmonary type. The younger the child the greater the predominance of the bovine type. From the standpoint of our present knowledge we must consider that practically every case of bovine tuberculosis in children is ingestion tuberculosis, contracted from milk or fresh milk products. Infants and young children whose only nourishment may come from tuberculous cows must ingest tubercle bacilli even several times a day for months.

Careful study of a series of 100 cases at Lakeville showed that 4% of the cases under ten years of age with tuberculosis of lymph glands and 16% of those with bone and joint tuberculosis in the same age group were due to infection with the bovine type of organism. Many authors report figures of their studies in this work which lead us to feel that our percentages are low as compared with many. They are sufficiently high, however, to make every one of us, who at any time have the responsibility of the care of children during the susceptible ages of the first ten years of life, feel our obligations very keenly as to the need of protecting every child from possible sources of infection from raw milk from an untested or reacting herd of cows. Our histories show that many of these children were fed with cow's milk during the early years of life.

The eradication of bovine tuberculosis which has been brought about during the past ten years has resulted in a vast improvement in the incidence of tuberculosis. This work must go on, however, and the medical profession should be the one to demand and insist upon its completion. If even a small number of these children can be protected from infection from this known source, think of the suffering and crippling that can be prevented, to say nothing of the economic side of the picture. If we would protect our own child we owe the same protection to every other child coming under our care or observation.

At the sanatorium an accredited herd of cattle is kept, sufficiently large to supply all the milk used in the institution. All milk is clarified and pasteurized and we feel that the importance of such control, where the majority of our patients are children already suffering from tuberculosis, cannot be over-estimated. The physician in the community can render great help by continuing his endeavors to prevent tuberculous infection from animal to man by advising and assisting mothers to obtain milk that is known to be safe for consumption by infants and children.

The human sources of infection can of course be best eliminated by isolation of the patient suffering from active pulmonary tuberculosis. Any individual in the community with expectoration containing tubercle bacilli is a potential source from whom other persons, especially children, may, and frequently do, become infected. It is the prevention of this sort of infection that has gone a great way toward the decline in the death rate from tuberculosis. One of the purposes of the sanatorium for pulmonary cases is to eliminate these infectious cases from the home. The education they receive in the sanatorium helps to prevent future breakdowns. As infections from so-called positive cases of pulmonary tuberculosis take place by sputum containing bacilli being transmitted from one individual to another or by articles contaminated by this sputum, the physician's duty with a known positive case is to eliminate the patient from the home containing children or so to educate the patient and his family that the danger from this source can be practically eliminated. It must be borne in mind, however, that the presence of an open case of pulmonary tuberculosis in the same home with small children is a source of great danger under any condition. Children are very susceptible during the early years of life and in families with any cases of close contact, infection almost always results. Sixty-eight children reveal in their histories the fact that they had definite contact with other tuberculous members of the family and were closely associated with them for varying periods of time. In five more cases the evidence was of a suspicious nature, but definite contact could not be proved. While these 68 developed active disease and came under our observation, think of the very large number who must have received the infection and may at any time develop the disease. Whatever may happen in later life, we

are perfectly certain that most primary infections take place in childhood.

Up to the present time but 40 cases of true congenital tuberculosis have been reported. Max Zarfl[2] points out that there are a series of observations which indicate that tuberculosis may pass from the tuberculous mother through the placenta and infect the fetus if the placenta has become infected. The infection of the fetus may occur during the early stage of intrauterine life. Baumgarten considers the placental hematogenous infection during or before birth as the most frequent form although infection may occur also by swallowing or inhaling the infected amniotic fluid. Pathologic studies have shown that in the majority of these cases the liver is primarily involved together with the adjacent lymph nodes. In some instances the bacilli pass through the liver, enter the general circulation and may involve any organ or cause a generalized miliary tuberculosis. Early and massive infection causes intrauterine death of the fetus or death shortly after birth. An interesting case is quoted "of an infant born to a 23 year old mother suffering from chronic tuberculosis. The infant was slightly premature, weighed 2,700 gm. and was immediately taken from the mother. Eight days later the child began to run an irregular fever, had a positive tuberculin reaction on the sixteenth day of life, and a toxin eruption. In three weeks visceral changes became apparent including a distended abdomen, enlargement of the liver, dilatation of the superficial veins of the abdomen and thorax. At four weeks there was a generalized lymph adenopathy and skin tuberculosis. Death occurred on the thirty-ninth day. Autopsy showed a tuberculous process generalized by way of blood and lymphatics. There were three large caseous and calcified foci in the liver. The portal and upper aortic lymph nodes were involved. The lungs were diffusely infected apparently by a hematogenous infection. The clinical and anatomic diagnosis was a placental-hematogenous tuberculosis of the hepatic type."

It is when the first infection strikes undernourished individuals and those with lowered resistance from other causes that it is of consequence. When the infected child is in a very impoverished state of nutrition then infection rapidly develops into disease. We must avoid early infection by the isolation of children from the "tuberculous" environment, and further by keeping them in a good state of nutrition, so that they are armed against the infection which sooner or later takes place. It must be considered, that in the primary focus there are living tubercle bacilli which are virulent certainly for several years if not for life. These organisms may, when the patient's resistance is lowered, particularly by malnutrition, spread out and develop active tuberculous processes. Latent tuberculosis should not be allowed to become active.

When a parent brings a child to the office for examination[3] because he knows that the child has been exposed to tuberculosis, we should be particularly careful in our examination and management, having in mind that the younger the child and the younger the baby the greater the danger of infection. The newborn, especially, should be guarded against tuberculosis. Some feel that it is not advisable for the newborn baby even to live in the same house with anyone known to have open tuberculosis. An open or suspected case of tuberculosis in the mother is an absolute contraindication to nursing, not alone because of the danger from the milk but because of the close contact between baby and mother. Ordinary precautions that will protect the adult or the older child are entirely inadequate for the baby. The baby that has been exposed even for a short time to an open case of tuberculosis and the older child that has been seriously exposed should be considered tuberculous and so far as possible treated as such, no matter what the present state of health may be. In these young children the Pirquet test is of great value especially if it is negative. While this test may be slightly less delicate than the intracutaneous test, the practical value of the difference is doubtful. The baby with a positive Pirquet test should be regarded as a tuberculous patient and if properly and promptly treated the prognosis is fairly good. In older children the positive test may mean little more than infection but is of considerable more importance than is a positive test in an adult.

Brennemann and Gittings both state that a positive tuberculin test in a child under two years of age is indicative of active tuberculosis. Our findings agree with these observations.

R. Kjer-Petersen and J. Ostenfeld[4] reported an interesting review of 293 cases with positive Pirquet reactions in the first three years of life, 209 from homes in which there was open tuberculosis and only 40 from homes free from sources of infection. Five years later, of the 53 patients with positive Pirquet reaction in the first year, 20 had died (nine before the end of the third year, 19 from tuberculosis) and 33 were well. Of the 91 in the second year 14 had died, 13 from tuberculosis, and 77 were well. Of the 149 in the third year 15 had died, 13 from tuberculosis and 134 were well, which goes to show that the longer the infection can be delayed the better the prognosis for the child.

In Massachusetts during the school year 1930-1931, out of 48,600 grade school children tested, 26% reacted to the Pirquet test: of 8,730 high school children 43% gave a positive tuberculin test. In the grade school group, 13 cases showed

evidence of pulmonary tuberculosis and 394 cases of hilum tuberculosis. In the high school group 19 cases showed evidence of pulmonary tuberculosis and 15 had hilum tuberculosis. In other words three per cent of the grade school children reacting to the Pirquet test were found to have hilum tuberculosis.

All children admitted to the sanatorium in this group were tuberculin tested by the Pirquet method, both human and bovine tuberculin being used. The percentage of reactors was as follows: human 7.75%, bovine 8.25%, and both human and bovine 84%. Patients negative to the Pirquet test, with both human and bovine tuberculin, were again tested by the Mantoux method, the strength of the tuberculin used ranging from 1-10,000 of a milligram up to a milligram. No positive reactions were obtained by this method in the Pirquet negative cases.

Teachers, nursemaids and governesses may transmit tuberculosis often enough to justify the checking of this source, as association, for only a few hours a day, with an open case of tuberculosis may infect and perhaps destroy the young infant or child. These nursemaids frequently contact children much more than parents do, and also other children in the community[5].

With school children the school itself may play a part in the dissemination of tuberculosis if it is overcrowded, poorly ventilated and not kept clean, has inadequate playgrounds and a curriculum that overtaxes the strength of the pupils.

Prevention of tuberculosis or any other disease requires the aid of the practicing physician who is really the ultimate health officer. As Sir James MacKenzie[6] so wisely said: "I . . . assert with confidence that medicine will make but halting progress while whole fields essential to the progress of medicine will remain unexplored, until the general practitioner takes his place as an investigator. The reason for this is that he has opportunities which no other worker possesses—opportunities which are necessary to the solution of problems essential to the advance of medicine."

Later Holt[7] noted the following conclusions: "I am impressed with the fact that there are certain phases of medicine, opportunities for the observation of which are furnished only by private practice. It is greatly to be regretted that those who are able to follow the same patients over long periods of time, and so are able to see ultimate results of acute disease, seldom keep records of their observations or if they do, seldom publish them. Such contributions would fill a large gap in medical literature, particularly in the matter of prognosis."

In examining the preschool child and those in the primary grades, the tuberculin test should be used freely. If a child reacting to tuberculosis is found, the rest of the family should be examined and checked up so far as is practical. If the source of infection can be found it should be eliminated. Other reacting children should be supervised and placed on a program to prevent the infection from becoming disease. The state of the child's nutrition, his height, weight, habits and the condition of his teeth and tonsils are very important.

In Massachusetts, for 1931, the deaths from pulmonary tuberculosis were 51.6 per 100,000. All other forms were 5.8 per 100,000. The decline in the death rate in children under ten years has been steady and encouraging, the drop by five-year periods being as follows:

1904-1908	34.0 per 100,000
1909-1913	22.8
1914-1918	18.5
1919-1923	11.4
1924-1928	6.5

The greatest decline in any age group has been in the group under ten years, but in spite of this decrease the fact remains that the greatest number of deaths from tuberculosis still occurs in the age group under five years.

In considering the early diagnosis of juvenile tuberculosis, we must bear in mind that physical signs are indefinite and often misleading. The history and symptoms are more important as a basis for treatment than physical signs. The x-ray is of the greatest aid in diagnosis.

X-ray examination of children's chests for primary calcium lesions calls for very careful study as shown by Miller's statement[8] that "With a technique of examination, based upon careful study, calcification of tuberculous first infection in the thorax is demonstrable roentgenographically in only about one-quarter of the existing cases. The superimposition of structures of varying density, the factor of cardiac and intrapulmonary vascular movement and the minute size of many of the calcium deposits are responsible for this. Blood vessels roentgenographed axially produce shadows which may be confused with those of calcium."

Opie[9] feels that cases of older children characterized by the following require no special prophylactic care: (1) Infection revealed by the tuberculin test with no lesion demonstrable by roentgenologic examination. (2) Circumscribed pulmonary nodules that have undergone calcification. (3) Roentgenologically demonstrable tuberculosis of tracheobronchial lymph nodes, with exceptions noted later.

Children with the following conditions should be given prophylactic care as they are in danger of developing clinical tuberculosis: (1) Latent tuberculous infiltration of the childhood type evident in roentgenograms, often associated with tuberculosis of tracheobronchial lymph nodes. In some instances, particularly in older

children, these lesions represent healed infection and do not require special care. (2) Tuberculosis of tracheobronchial lymph nodes with some calcification, recognizable by roentgenologic examination, (a) when the child is still in contact with open tuberculosis, (b) when the tuberculin reaction is intense, (c) when the lesions are unusually large or very numerous. (3) Latent tuberculosis of adult type. (4) Arrested pulmonary tuberculosis, notably in children who have been discharged from sanatoriums. (5) An active tuberculin reaction or an otherwise negligible tuberculous reaction recognized by roentgenologic examination, when associated with impaired health and conspicuous underweight, which may not be referable to tuberculosis.

Sanatorium treatment or its equivalent should be given children who show: (1) Tuberculous lesions accompanied by symptoms and physical signs referable to tuberculosis. (2) Latent lesions of the lung of childhood or adult type that on repeated examinations are found to be progressive. (3) Massive tuberculosis of tracheobronchial lymph nodes that has not undergone calcification.

Observations show that weight below normal is not found more frequently in children who react to tuberculin than in those who do not. Many children with recognizable tracheobronchial tuberculosis are considerably above normal weight. In children with the adult type of pulmonary tuberculosis and active extrapulmonary lesions, weights below normal are the rule.

The practical solution of the problem of tuberculosis in adults depends in a large measure on the attention that is given children with latent or active tuberculosis. Especially does this apply to the various forms of tuberculosis in the spine, hip and knee or for that matter any of the weight-bearing structures. Many patients give the interesting history of tuberculous lesions of recent development which without question have metastasized from an early lesion first demonstrated in childhood but not completely healed at that time. The cases of spinal caries with marked kyphosis, in some instances even to acute angulation of the spine, are painful examples of the progress, slow but sure, of incompletely arrested and unhealed processes. Some come in with the history of having had hip disease at five and now a knee or spine plus the old hip at twenty. Careful history reveals the story that the hip lesion was treated for a short time and as soon as the acute symptoms subsided the patient was permitted to resume his or her former activities without supervision for years. In view of our observations we feel now that these tuberculous joints should be fused as soon as the signs of acute disease have subsided and signs of healing by nature's methods can be demonstrated.

For years the teaching in medical schools was that tuberculosis in children during the first two years of life was always a generalized process. We have had many demonstrations contrary to this theory. We have seen many children under two years localize their lesions in the spine, hip, knee, glands and other structures of the body as well as limit their disease to the small bones of the hands and feet. The prognosis in the majority of these children, in the absence of other underlying conditions, is very often good. Prolonged intensive treatment is always required, however.

Routine examinations of the chest, including x-rays, have revealed varying conditions in this group of children. Twenty-nine revealed hilum tuberculosis and nine showed the process to have extended out into the parenchyma of the lung.

The non-tuberculous pulmonary conditions encountered were bronchopneumonia, bronchiectasis and lung abscess. Careful history and study of these cases generally result in a correct diagnosis. Bronchoscopic treatment in cases of bronchiectasis and lung abscess, draining through a bronchus, produce remarkable results and in many instances prompt cures.

Persistent low-grade temperatures in the absence of signs of an active lesion are frequently very difficult to diagnose. A negative tuberculin test in these cases is of considerable value in ruling out tuberculous conditions even though it does not aid in leading us to a definite diagnosis.

Without careful observation and examination, an unresolved pneumonia may result in confusing the diagnosis with tuberculosis. But one must in the presence of a positive tuberculin test exclude a possible tuberculosis. The x-ray findings assist in no small measure in these cases, but we must not expect the x-ray alone to make the diagnosis. We have seen a unilateral bronchopneumonia diagnosed as tuberculosis clear up as promptly as we would expect it to do and the prompt disappearance of the clinical and x-ray findings of course ruled out tuberculosis. In these cases the x-ray can be nonconfirmatory as well as confirmatory.

Some of the extrapulmonary conditions confused with tuberculosis were Legg Perthe's disease, arthritis, congenital hip conditions, renal diseases, intestinal diseases, blepharitis and conjunctivitis, and non-tuberculous adenitis. In children under ten years of age with positive Pirquet reactions, it is often much easier to call the above conditions tuberculous, unless the child can be kept under observation long enough to rule out tuberculosis, than it is to determine the real cause of the disease. It is not unusual for us to find it necessary to keep a child under observation and study for from 30 to 90 days

before we feel justified in arriving at some definite conclusions.

The idea that the x-ray is a final instrument of precision in the diagnosis of tuberculosis, especially of the chest, is not wholly restricted to the laity. The roentgenologist is expected to render an opinion and that opinion becomes for all practical purposes a diagnosis, whether he intends it to be so. We are all familiar with the roentgenologist who has a veritable tuberculosis complex. Every hilum shadow that is denser or more extensive than an assumed normal, every area that is a little more mottled than is usual, leads him into a suspicion of tuberculosis, and in tuberculosis an expressed suspicion soon becomes a certainty. We must put the whole picture together before we attempt to name it. We may have a superimposed tuberculosis in any of the simulating nontuberculous conditions and must therefore have it in mind.

Another conspicuous finding in this group of children was the fact that 28% on admission were below what we considered as well nourished and approximately half of this 28% group were definitely suffering from malnutrition in some degree. Now the question arises, did the malnutrition precede the active tuberculous disease and thus constitute an exciting factor or was it a secondary condition to the disease itself? The parent's story helps in getting the answer to this question. Four children had positive signs of rickets. If lowered resistance can be considered as a bridge between infection and disease the above factors cannot be dismissed as unimportant.

Malnutrition must be considered seriously and not as just one of those conditions that can be treated lightly or disregarded entirely so long as the child is not actually sick. The diagnosis of malnutrition, however, should be made only after careful examination by the physician and not alone by the height and weight chart.

One should hesitate to accept the many reports that this child is so many per cent underweight and that child is a certain per cent overweight. To do so we must accept one type of body build as normal for the whole nation. This is of course not so.

The malnutrition resulting from anorexia, which is found in the home and can be explained on a psychologic basis, must be considered as one of the definite causes of lowered resistance and one of the exciting factors in childhood tuberculosis. It is a very common occurrence for a parent, generally the mother, to bring the patient to the sanatorium for admission and we are promptly informed that the child cannot, does not, or will not eat this, that or the other article of food. The child usually has the appearance of a poor feeder. Unless the child is very toxic or suffering from an overwhelming infection, within a week or ten days following admission he is eating the usual house diet which in many instances contains some or all of the articles of food which he has previously refused at home. In other words except as a symptom of an active diseased condition, anorexia is practically unknown in the sanatorium. Similar observations are reported from institutions of other kinds. These children in the home seem to rebel naturally against the oversupervised and strictly regulated diet.

In older children the problem of anorexia in recent years has been somewhat increased by the intensive campaign of weighing, measuring and examining children and trying to bring them all to one standard by prescribing certain food elements, especially as to quantities, percentages, calories, calcium and iron contents, to say nothing of vitamins.

The diet of the older child can be and in several instances has been as confusing and unappetizing as the complicated formulas of the infant. Can we regulate with precision the amount of food per day and per feeding without considering the individual's characteristics and requirements?

As one pediatrician recently stated: "Children are individuals, physical as well as mental. The greyhound and the airedale are as healthy, normal and efficient as the mastiff and the St. Bernard. Some children are 'skinny' because they are active, and active because they are 'skinny'; others are phlegmatic because they are fat or fat because they are phlegmatic. There is no reason in health, other things being equal, why one should be converted into the other or why the two should be brought to a common artificially determined medium. I think it is a well-grounded pediatric conviction that the thinner, active child is a better health risk than the fat, phlegmatic child and, at least, an equal risk with the so-called normal weight child. The thin, active child with a good appetite and a good color gets, but hardly requires, a thorough physical examination. His very activity and lack of fatigue exclude a clinical tuberculosis. The child, on the other hand, that is thin and run down, has a poor color and appetite, tires quickly and is manifestly sick, requires the most careful scrutiny as to a possible tuberculosis, no matter what other abnormalities he may present."

It cannot be emphasized too strongly, on the other hand, that a normal weight and an apparently normal state of nutrition do not exclude tuberculosis. The baby with an extensive pulmonary tuberculosis may be of average weight, may even gain normally for weeks and months up to that inevitable rapid loss as the end approaches. It is a melancholy and depressing fact that the child, young or old, that has a tuberculous meningitis, the result of a sudden hematog-

enous invasion from a previously insignificant and unrecognizable lesion, is as often as not an otherwise normal physical specimen. Much good, however, has been accomplished by these examinations in bringing to light some of the causes of malnutrition that we can correct, such as faulty health habits, physical defects, especially enlarged or infected tonsils, adenoids, deviated septa and sinus infection, chronic otitis media, carious teeth, eye-strain, chronic over-fatigue, nervous excitement in or out of school, too much home work, movies, too many outside activities, either mental or social, lack of fresh air and sunshine, too little rest and sleep, and faulty dietary habits. Acute diseases are of course a cause but they require more than we can frequently give to prevent. While poverty is doubtless a cause, parental ignorance and shiftlessness are much larger contributing factors. Many children in good homes are unfortunately malnourished.

Retardation may be caused by malnutrition and physical defects of disease, such as tuberculosis, diseases of bones, and deformities. Diseases of the eyes, errors of refraction, diseased tonsils, adenoids and teeth are also often the basis of backwardness. The earlier the onset and the severer the degree of impairment, the greater the retardation. Retardation is usually a functional rather than an organic defect.

The teacher, who is a keen observer, can quite accurately determine the improvement in a child's condition by the progress he makes in his studies. The accomplishments of the child of normal mentality, as soon as he is free to gain and his resistance is raised so that he in turn overcomes diseased conditions are, for a period of time, greater than those of the average child in classes with children of his own age and grade. Intelligence tests on many of these children on admission classify the child as one or two years below what is considered the average for his age. Re-tests later, after a lapse of from one to three years, during which time the child has shown good improvement toward recovery from his disease, and has received instruction in school classes, reveal the fact that the child's rating is now up to that which is considered normal for his age and in some cases even above the standard for that age group.

We cannot expect the child who is physically handicapped to continue the development of his mental faculties so rapidly as those free from such handicaps.

In chronic fatigue the symptoms may be physical or mental in character. The fatigued child suffering from the effects of mental and physical overstrain becomes malnourished and nervous. Nervousness, fatigue and malnutrition are not separate entities but syndromes of the same process of functional disorders. Children with bad habits, especially those of eating, frequently become irritable and restless and finally suffer a loss of weight.

Many of these children require less activity rather than forced feeding with high fats, etc., and it is this emphasis on rest rather than extra food that is frequently the keynote of success.

It has always been the aim of the sanatorium to have physical defects corrected as fully as possible immediately after admission so that the children may be free to gain. without obvious physical handicaps. It is necessary on many patients to do large amounts of correctional work. Dental care and removal of diseased tonsils and adenoids have constituted the major part of the correctional work. The wholesale removal of tonsils has never been approved, but where there is fair reason for believing that the condition of the tonsils or adenoids is definitely interfering with the child's well-being the Staff has felt justified in recommending surgical measures. Four or five per cent of the removed tonsils and adenoids have shown tuberculous infection, but we have every reason to believe the true number of infections is considerably higher.

Routine Wassermann tests reveal an occasional case of syphilis. The importance of treating this condition needs no further mention here.

Much has been written and said pro and con in regard to the immunization of children against various contagious diseases. Bearing in mind that we are observing a group of children suffering from a general constitutional disease, I feel that the immunization program that these children have gone through will furnish the answer to some, at least, of the theories advanced each year as to the contraindications for vaccination, and the administration of toxin-antitoxin for diphtheria.

Every child admitted to Lakeville regardless of age is vaccinated and immunized against diphtheria and we have thus far been unable to demonstrate any ill-effects or untoward reactions. The methods and technique are those recommended by the State Department of Public Health. Temperature, pulse and urinalysis are carefully checked on this entire group on the days following the injection of the toxin-antitoxin for diphtheria and no toxic reactions have been noted. The incidence of contagious diseases has been very low and limited to chicken pox, pertussis and an occasional case of mumps. They all recovered without complications.

The mouth and its adnexa have many important functions in the preservation of health and in quite the same degree present possible relationships to morbid processes too numerous to mention. The mouths of children upon admission reveal many interesting pictures. A majority present a low-grade gingivitis. Most of these cases clear up with the improvement of the child's general condition and without local treatment. The color of the gums changes from the red, inflamed, soft bleeding type to the nor-

mal pink, firm non-bleeding type. Every attempt is made to retain as long as possible the deciduous teeth which are so important in determining the eruption of the permanent dentition and the growth and development of the dental arches.

On the initial examination extensive caries of the deciduous teeth is frequently found. Many of these are condemned and charted for extraction at the first examination, but it is frequently possible to retain a large number of these teeth until the proper time for exfoliation, by the arrest of the carious process which takes place during the early months of treatment as shown by the formation of a very hard resistant layer of secondary dentin to replace the original soft leathery decay. Fresh air, sunlight, orange juice and cod liver oil all play an important part in the arrest and repair of the dental structures. In many cases wherein the pulps of these teeth were not actually exposed by the carious process, the aid of dental treatment was purposely omitted to prove the above statement. Some doubtful cases are aided by the silver reduction process consisting of ammoniacal silver nitrate and formalin.

Children who have been in the sanatorium for a long period of time and appear periodically for reëxamination and prophylaxis almost always present a dental condition requiring very little extensive work such as large fillings or extractions because of caries. Their teeth will present a characteristic black stain which forms around the cervical margins, and which, although several theories have been advanced, in our experience, has always been associated with a good dentition.

These children are not afraid to visit the dentist; they come willingly, are seen often, and consequently have not the dental wrecks which some outside children present. They are well drilled and carry out an efficient oral hygiene program.

The first dental examination of the child should be made as soon as the deciduous molars are erupted. This takes place between the ages of two and two and one-half years. Defects have been noticed even earlier than this and it is important to correct them as soon as they are evident. If there are no defects the teeth should be cleaned. Reëxamination and prophylaxis should be routine every three months until the age of five and then every six months or one year as necessary until the second dentition is complete. All early cases of deformity of the jaws or malocclusion should receive immediate attention.

The teaching of dental hygiene should be a program indulged in by the mother, the teacher, the physician, and the dentist. The mother's duty is to teach the child cleanliness, the use of the tooth brush and mouth wash, to take him to the dentist periodically and to make the visit a pleasant one. There are no nerve structures in the enamel of the teeth, therefore no pain or other sensation at the onset of decay and these beginnings can only be detected by examination. There is consequently less discomfort from treatment and less likelihood of subsequent disease if it is done at this time. It is our duty to see that the child is taught the value of good sound straight teeth for use, appearance and health. The child should be further taught that good teeth can be made and preserved only from the right materials derived from the proper food and feeding and the practice of other health habits essential for good nutrition; that teeth are living structures and depend for their health upon the body in general which in turn is affected by diseased teeth. It is the duty of the physician to instruct the mother in the fundamentals of diet and nutrition in relation to the teeth of her child from the very time of conception, for the formation of teeth begins long before birth; in fact the calcification of the deciduous dentition is already completed at birth. Therefore the physician should be concerned with the health habits of the mother and the quality and quantity of food materials she furnishes to the child during pregnancy. He should instruct her in the feeding, sunning, airing, sleeping, exercising, and clothing of the child after birth. He should follow the condition of the child as regards dental possibilities up to the time the child makes its contact with the dentist, and if necessary report earlier any infirmities or defects with the advice to consult a dentist. The diet and nutrition of the child are always the responsibility of the physician. For instance if he notices the clinical picture of scurvy, he should supplement the diet accordingly. If he detects erosion of the enamel of the teeth, he should prescribe the necessary vitamin D content to be added to the diet.

M. Mellanby and C. L. Pattison give an interesting account of the action of vitamin D in preventing spread and promoting arrest of caries in children as follows: In a group of 21 children whose average age was about five and one-half years, the addition of vitamin D checked the initiation of new carious points and arrested the infective process in many carious teeth. This investigation corroborates those previously made in which it was found that the carious process in the teeth of children could often be inhibited by increasing the intake of fat-soluble vitamins by the addition to the diet of cod liver oil, egg yolk and extra milk. The present work shows that vitamin D is a most powerful agent in these inhibitory processes.

It is the duty of the dentist to teach that 90% of children have, on their first appearance, imperfections of enamel in which decay will probably begin unless they are treated. The earlier decaying teeth are treated, the more certain will be their preservation. Crooked teeth look badly, they affect the general health and much

can be done toward their improvement, consequently he should examine and clean the teeth periodically and repair all defects. He should teach the child to take an interest in his own teeth and to assist the dentist in maintaining a clean, good-looking, efficient oral machine. Many of our defective teeth come from racial and parental ignorance on this subject, and more progress can be made by educational methods by the physician than in any other way.

I shall not enter into the field of surgery in this paper other than to say that our results show that surgery plays as important a part in the treatment of children as it does in adults. Except in cases where there is an acute surgical condition, intensive medical treatment should generally precede surgery. Satisfactory treatment of diseases in children requires the closest coöperation between the medical man, the surgeon, and physicians in the various specialized fields of medicine. Most satisfactory results in the treatment of any abscesses in children have been obtained by waiting until they were practically ready to rupture before opening. This insures good drainage and prompt healing. If there is a tendency to recede they are best left alone.

In the matter of medical treatment our energies have been directed toward procedures known to aid rather than interfere with nature's methods of cure.

Rest, food, and air stand out in the treatment of non-tuberculous conditions as well as those with a tuberculous origin, especially when located in the upper respiratory tract. Of these three agents there is no longer any doubt that rest, rest in bed, complete rest, whenever there is fever and fatigue, is the greatest single general therapeutic agent; and it is just as effective and just as easily carried out in the child as in the adult. Regardless of the disease, rest is the greatest natural medical ally. The child must of course eat if we expect recovery. In tuberculosis, and it is equally true in other conditions, the child with fever will eat better if he is at rest in bed. Many will eat much better in a different environment than at home, and frequently better for a nurse or some other stranger than for the mother. It is useless to coax or try to force a child to eat. Cod liver oil has a distinct value as a food and is apparently well tolerated by all children in one form or another. Orange and tomato juices should be included in the daily diet. There is no substitute for fresh air. Out-of-door air should be used generously day and night, winter and summer. While in the open air the sun's rays judiciously used have a very beneficial effect on the human body. Artificial heliotherapy indoors is but a substitute at best. Exposures should not be carried out so enthusiastically that too great a protective mechanism is built up in the form of deep pigmentation, which may act as an insulating rather than a transmitting medium.

We have encountered rickets in varying degrees and the response to ultraviolet light both natural and artificial plus the use of cod liver oil have promptly and effectively corrected even the most marked cases. The complete suppression of all signs of rickets during the first year of life, however, may be accomplished only through intensive treatment begun almost from birth and continued regularly at least throughout the first year.

Observations by Wyman and Drinker and Mackenzie[10] have shown that there are sufficient short rays in the sun's spectrum in Boston (L. 42° 21′) in the winter to be of use in the prevention and cure of rickets in children, even when passed through different types of ultraviolet transmitting glasses. An analysis of the results of their study shows:

1. The infants exposed during the months of November, December, January, and February become deeply tanned. Those treated after March 1, however, became more quickly tanned.

2. In the majority of the cases, beginning within two weeks of exposure, there was a sustained rise in the phosphorus-calcium ratio.

3. Deposition of calcium, as shown by the roentgenograms, could be seen following exposure after the second week in all of the cases, with the calcification continuing to increase throughout the period of observation.

The hope of the eradication of many of the diseases of our children continues to lie in the prevention rather than in the cure.

To quote President Hoover[11] "The ideal to which we should strive is that there shall be no child in America:

> That has not been born under proper conditions;
> That does not live in hygienic surroundings;
> That ever suffers from undernourishment;
> That does not have prompt and efficient medical attention and inspection;
> That does not receive primary instruction in the elements of hygiene and good health;
> That has not the complete birthright of a sound mind in a sound body;
> That has not the encouragement to express in fullest measure the spirit within which is the final endowment of every human being."

At the dedication of a children's building at Lakeville a few years ago, Dr. Otis in his remarks told a little story that it seems to me is well worth repeating. The moral I would leave with you tonight. "There was a curving road around a high bluff, and many travelers fell over it and were injured. Finally the accidents became so numerous that the citizens of the community called a public meeting for the purpose of raising funds for the erection of a hospital to

take care of the injured persons. During the discussion of ways and means one hard-headed citizen suggested that they build a stout fence around the road on the bluff and then there would be no need for a hospital."

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GRANTS BY THE MILBANK MEMORIAL FUND

The question of how to increase the purchasing power of the public health dollar will receive renewed emphasis in the policy of the Milbank Memorial Fund, which granted $843,337 last year for philanthropic projects, according to its annual report published recently.

In the twenty-seven years since it was founded by Mrs. Elizabeth Milbank Anderson, this Fund, of which John A. Kingsbury is the secretary, has appropriated $8,987,575 for public health, social welfare, and education. During that period 147 projects have been aided. Last year its grants amounted to $843,337. Because of emergency conditions in New York City the directors of the foundation, as in the preceding year, drew on capital to supplement its gifts out of income. It appropriated $300,000 in 1931 for use by the Emergency Relief Committee and $50,000 to the Women's Fund Committee of the same organization. The main interest of the foundation, however, is in the field of public health.

During the past year, according to Mr. Kingsbury, the emphasis of the Fund has shifted "from experiments in applying commonly accepted procedures, as in the New York health demonstrations, to experiments with new or improved public health administrative methods, these methods themselves being the outgrowth of analysis of past experience and planned especially for community programs". In view of present-day economic problems the report stresses "the need of obtaining the greatest efficiency in public health work, in order thereby to increase the purchasing power of the public health dollar, whether that dollar comes from taxation or from voluntary contributions".

Projects which have been given financial assistance by the Milbank Fund are as follows: The Committee on the Costs of Medical Care; the New York State Health Commission, appointed by Governor Franklin D. Roosevelt in 1930; the New York Health Demonstrations; Health activity in the Bellevue-Yorkville Health Center, New York City; and the New York Diphtheria Prevention Commission.

Other organizations in New York City aided by the Fund last year are the East Harlem Health Center, East Harlem Nursing and Health Service, Inc., Judson Health Center, and the New York Principals Association Committee on Health. A grant was made to Syracuse University in support of a course in public health nursing.

Milbank contributions toward social welfare and relief include a grant to the New York Association for Improving the Condition of the Poor, and a payment of the Fund's capital on its pledges of $500,000 toward endowment for the State Charities Aid Association.

Besides carrying on research by its own staff the Milbank Memorial Fund has granted aid to various other research projects. It is coöperating with the United States Public Health Service in a study of public health methods in two counties of Virginia. It is also collaborating with the United States Public Health Service in clinical research on syphilis and in epidemiological studies in Cattaraugus County. The Fund last year granted $7,500 for research in the laboratories of the New York City Department of Health on the value of the Bacillus Calmette-Guérin as a vaccine against tuberculosis. A grant was made to the Manhattan Eye, Ear, and Throat Hospital, for research, and a payment to the Edward L. Trudeau Foundation on a previous grant for tuberculosis research.

Population problems involving health conditions and activities are being investigated by the Fund. These include a study of differential fertility according to social class and a study of the prevalence and effectiveness of methods of family limitation. The latter project is directed by Professor Raymond Pearl of The Johns Hopkins University.

A distant beneficiary of Milbank philanthropy is the rural health center in Ting Hsien, China, the first rural health demonstration in that nation.

The Milbank foundation's division of publication reports the issuing of five volumes during 1931 in coöperation with publishers for the book trade, namely, "Health on the Farm and in the Village," by Professor C.-E. A. Winslow, a critical survey of the Cattaraugus County Health Demonstration; "School Ventilation, Principles and Practices," by the New York Commission on Ventilation; and a series of three volumes by Sir Arthur Newsholme entitled "International Studies on the Relation between the Private and Official Practice of Medicine with Special Reference to the Prevention of Disease."

The president of the Milbank Memorial Fund is Albert G. Milbank. Other members of the Board of Directors are Edward W. Sheldon, Thomas Cochran, Frank L. Polk, Linsly R. Williams, M.D., Roland S. Morris, and Cornelius N. Bliss.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18461

AN UNUSUALLY RAPID COURSE IN PULMONARY HEMORRHAGE AND PLEURAL EFFUSION

PRESENTATION OF CASE

DR. DAVID A. YOUNG*: This was a forty-nine year old Italian laborer who was first seen by Dr. Cass in the Outpatient Department a month before admission. At that time the patient gave a history of chronic cough for a year which had recurred four months before the visit. It had been accompanied by sputum. Two months and a half and again two days before the visit he coughed up blood. Examination showed rhonchi in his chest and a friction rub; apparently no localizing signs except a little dullness on the left side. His sputum was negative for tubercle bacilli by the Ziehl-Neelsen stain. At another visit six days before entry he was seen by Dr. Urmy, who found flatness to dullness at the left base with diminished tactile fremitus, some egophony and diminished to absent breath sounds. The whole story goes back to cough with sputum a year before entry for two months; then five months before entry cough with some white to orange sputum with foul odor, with some pain in his left chest on coughing, the cough and pain persisting until his admission. Three and a half months before entry he had a cold in his chest and coughed up about 60 cubic centimeters of blood. A month before entry he coughed up half a pint of bright red foamy blood. At that time he had edema of the ankles, dyspnea on exertion and orthopnea. One week before admission he raised more blood, this time bright red but not foamy. During the past month or two he had been living mostly on alcohol. He had had a weight loss of eighteen pounds in the past few months.

The past history is of no help except that he had had a Neisser infection fifteen years before entry and occasional nocturia.

On physical examination he was fairly well developed and nourished, with dark skin, a considerable amount of hair and brown pigmentation of the sclerae. Chest examination showed signs of fluid in the left chest, with the heart slightly displaced to the right. By percussion the heart was within normal limits. There was curving of the finger nails but no definite clubbing. There was marked edema of the feet and ankles, slight edema of the shins.

Blood examination showed a hemoglobin of 45 per cent, red cells 4 million. The white cell count varied from 18,000 to 21,000. There was a predominance of polymorphonuclears, some achromia. The platelets were diminished. The sputum showed no tubercle bacilli, rare spirochetes.

A PHYSICIAN: Was there an x-ray note?

DR. TRACY B. MALLORY: Dr. Martin's note on the first plate was: "The right diaphragm, costophrenic angle and lung field show no definite variation from normal. The left diaphragm shows slight limitation of respiratory motion. The left midlung is distinctly abnormal and shows a dense pyramidal shadow the apex of which is directed towards the pulmonary conus, the base of the pyramid lying along the lateral axillary border. At its upper edge there is a rounded 2 centimeter area of decreased density which might be a fluid level, not definitely visualized in the oblique view. The dense shadow in the left lung might be in the region of the interlobar septum."

The report on the next plate, which was taken almost a month later, is: "The appearance of the left lung shows some increase since the last examination. This increase in the size of the lesion has occurred after tapping. There is no displacement of the heart or mediastinum. The diaphragm and costophrenic angles are now obliterated by a dense homogeneous shadow. There is still a questionable fluid level at the upper level of the homogeneous shadow."

They did not commit themselves to any diagnosis.

DR. YOUNG: The patient was seen in the Thoracic Clinic, where dullness, much diminished breathing, egophony and practically absent tactile fremitus were found at the left base. A chest tap gave 900 cubic centimeters of clear yellow fluid with a specific gravity of 1.018. There were 1000 lymphocytes, 83 per cent mononuclears and 17 per cent polymorphonuclears. Culture showed no growth. The chest tap was repeated and 500 cubic centimeters more withdrawn. It was decided that he probably would have to have bronchoscopic examination, but on account of his anemia that he should have a transfusion before that time. The donors were ready, when he suddenly began to cough up blood again. Within two days he had severe hemoptysis and a week after entry died.

CLINICAL DISCUSSION

DR. YOUNG: The impression in the Outpatient Department six days before entry was left pleural effusion and suggested malignancy. The impression on admission to the wards was possible tuberculosis or a suppurative process and perhaps a neoplasm. The visiting physician at that time thought it was a tuberculous process, but

*Senior interne on the East Medical service.

later favored neoplasm. The final clinical diagnoses were lung abscess, questionable carcinoma of the lung, secondary anemia and pulmonary hemorrhage.

DR. THOMAS V. URMY: I saw this patient only once in the Outpatient Department. I thought he had fluid in the left side of his chest and advised him to come into the hospital. He apparently did not come in at once. My impression was that he might have neoplasm.

DR. WYMAN RICHARDSON: The prominent thing about this very ill patient was a marked hemoptysis with only a moderate amount of sputum, not foul. He was obviously very sick. My feeling after I had seen him for a while was that he had malignant disease.

DR. FREDERICK T. LORD: With respect to the differential diagnosis a constant difficulty arises in patients with this complex of symptoms. Hemoptysis "out of a clear sky" is most likely to be due to tuberculosis and the exceptions to this are rare. In our experience malignant disease arising in the bronchi occupies second place as a cause of hemoptysis as an initial symptom. Lung abscess is not a cause in this group. In a small group of cases lung abscess arises secondary to malignant disease in consequence of the trapping of infection in parts of the lung supplied by a partly occluded bronchus or from necrosis of a tumor mass in the lung itself. The distinction between uncomplicated abscess and abscess secondary to malignancy is difficult. In this case the x-ray furnishes the most important evidence and in this there is very little to suggest tuberculosis or malignancy. I should think the chances in favor of uncomplicated abscess.

DR. EDWARD D. CHURCHILL: I do not remember seeing this patient, but should lean towards the diagnosis of malignant disease rather than of simple abscess.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Pulmonary abscess.
Carcinoma of the lung?
Secondary anemia.
Pulmonary hemorrhage.

ANATOMIC DIAGNOSES

Pulmonary abscess, left, B. pyocyaneus.
Septicemia, B. pyocyaneus.
Hydrothorax, left.
Ventral hernia.

PATHOLOGIC DISCUSSION

DR. MALLORY: I do not believe that it would have been possible to make on the data at hand a definite differential diagnosis between abscess and malignancy in this case. At autopsy it proved to be abscess only. There was a large abscess 10-12 centimeters in diameter, chiefly in the lower lobe but also involving the lower part of the upper lobe. The interlobar septum was definitely perforated at one point; elsewhere it was thickened up to 2 or 3 centimeters. The abscess had perforated through the pleura and there was a small empyema cavity about 4 centimeters in diameter.

One somewhat unusual feature of the case was that these abscesses when they were broken into at the autopsy table contained brilliant green pus. One often calls pus green, but one does not often see it really bright green as this was. On culture of the pus and also of the heart's blood we grew bacillus pyocyaneus in pure culture. It is an organism of relatively low virulence, but it can, not extremely rarely, cause a serious pathologic condition and even death.

DR. LORD: Where was the abscess cavity in relation to that apparent fluid level in the x-ray?

DR. MALLORY: I think that line is probably that very much thickened septum.

DR. LORD: What is the relation of this abscess to the pleura?

DR. MALLORY: It came right out of the pleura and at one point it perforated it. How old the perforation was I do not know. I think it had already reached the pleura when this second plate was taken.

DR. CHURCHILL: Were the bronchial glands enlarged?

DR. MALLORY: They were not noticeably enlarged.

DR. RICHARDSON: Was there anything else in the abdomen? There was some question of trouble in the abdominal region on account of the marked edema.

DR. MALLORY: No, we found nothing in the abdomen except an umbilical hernia. The heart also was negative.

CASE 18462

A CASE OF CHRONIC COUGH AND PROGRESSIVE EMACIATION WITH DEVELOPMENT OF FOUL SPUTUM

PRESENTATION OF CASE

DR. WALTER E. GARREY*: This is the case of a fifty-nine year old New Hampshire farmer of Scotch birth who was sent in from another hospital complaining of cough and general weakness.

He was in good health until thirteen months prior to entry, when he began to cough and raise several teaspoonfuls of white and yellow sputum daily. He lost twenty-five pounds in weight during the next four months. He was not incapacitated until nine months before entry, when he developed fever and was admitted to a hospital. A diagnosis of pleural pneumonia with effusion was made and a diagnostic chest tap was done which showed clear fluid according to a letter from the hospital, but no further details

*Interne on the East Surgical service.

as to the nature of the fluid were given. He gradually improved and was sent home two weeks after entry.

He gained eight pounds in weight and did light farm work until three months before admission, though he coughed persistently, mostly in the morning. The sputum was white, not foul, a cupful a day. He complained of a tender spot in the right chest just beneath the nipple. During this time his temperature was taken occasionally and was said always to be normal. Three sputum examinations were negative for tuberculosis. Four weeks before entry he seemed worse, felt sick and went to bed. His sputum began to be brown and foul and to increase in quantity. For two weeks before his admission to the Massachusetts General Hospital he had been in the hospital of his previous stay. He was referred to Dr. Wyman Whittemore here for possible surgical intervention. The trip down was made in the back seat of an ordinary closed automobile.

Examination showed an emaciated, very sick elderly man sitting up in bed and breathing rapidly. The mucous membranes were pale, the lips and nails slightly cyanotic. The left pupil was smaller than the right and reacted poorly to light. The tongue was dry and caked. The chest was flat and the ribs very prominent. There was an area of flatness at the base of the right lung with absent breath sounds and tactile fremitus. In the right axilla there was an area of dullness with diminished breath sounds and tactile fremitus. All breath and voice sounds were less in the right chest than in the left. Over the entire left lower lung were numerous coarse moist râles. The breath sounds were somewhat bronchovesicular. There was some curving of the finger nails, suggesting early clubbing.

The temperature was 99°, the pulse 110, the respiration 25.

The x-ray report is as follows:

"There is extensive dullness throughout the right lung field, quite intense in the lower portion of the chest, which ends in a sharp upper border resembling a fluid level opposite the fifth rib in the axillary line. Within the dullness both in the upper and lower chest there are numerous rounded areas of diminished density having the appearance of cavities. The diaphragm on the right is irregular in outline, the costophrenic angle obliterated. At the left base there is diffuse mottling along the course of the lung markings. The upper portion of the left lung field is clear. The heart shadow is in the midline. The trachea shows slight displacement to the right."

The evening of entry he coughed up a few drams of foul sputum. The next morning he was much more cyanotic and dyspneic and much more ill. The temperature was 101° rectal, the respiration 35, the white cell count 28,000, the red blood count 4 million, the hemoglobin 70 per cent. He was transferred to the Medical service. Râles were heard all over the lung fields. He went downhill, the respirations rose to 45, he became irrational and irresponsive and died at midnight the day after entry.

Clinical Discussion

Dr. Garrey: The impression on the Surgical service was that he had chronic bronchopulmonary suppuration, fluid at the right base, probably an active bronchopneumonia in the left lower lung. The question of tuberculosis or neoplasm was discussed at the time the chest plate was interpreted.

Note by Dr. Aubrey O. Hampton: On the film taken in this hospital there is nothing to suggest anything more than a destructive lesion involving the greater portion of the right lung, multiple abscess cavities and involvement of the pleura. There is a sharp horizontal line in the midportion of the right lung with air above it. A fluid level within an abscess cavity or within the pleural cavity could produce this picture. Films taken in various positions are necessary to prove the presence of a fluid level, and these are not available. The mottled dullness at the left base could be due to bronchi filled with secretions. There are also two or three small round sharply defined areas of density which could be beginning abscesses or metastatic tumor nodules.

Dr. Frederick T. Lord: This case presents much the same difficulty in diagnosis as the preceding, 11461. I remember the problem as it was presented at the Thoracic Clinic. The patient was almost in extremis. We thought he had a bronchopneumonia at the left base and a lung abscess at the right base. At the time we considered the problem an earlier x-ray of the chest was at hand and from the presence in this of a dense, sharply circumscribed shadow on the right side the possibility of malignant disease was suggested.

Clinical diagnoses (from hospital record)

Lung abscess.
Bronchopneumonia.

Anatomic diagnoses

Carcinoma of the right lung, bronchiogenic.
Multiple pulmonary abscesses.
Bronchopneumonia, left lower lobe.
Arteriosclerosis, aortic and renal.
Cortical cyst of the left kidney.
Hypertrophy of the heart.

Pathologic Discussion

Dr. Tracy B. Mallory: This case turned out at autopsy to be a cancer. We have the specimen here. It is an exceptionally characteristic one. Here is the primary bronchus and projecting up into it, completely filling the bronchus to the lower lobe and more or less occluding

the bronchus to the upper lobe, is this finger-like mass of neoplastic tissue.

A SURGEON: How long is that, Dr. Mallory?

DR. MALLORY: It is 2.5 to 3 centimeters from the attached base to the apex of the tumor, which is in the bifurcation.

The tumor has invaded the lower lobe massively, coming out within a centimeter of the pleura. I think the sharp line that you see in the x-ray is the upper border of this tumor mass.

In the lower lobe of the lung on the opposite side there was a bronchopneumonia with two small abscesses each about two centimeters in diameter. The more common thing when we have abscess associated with malignancy is to find a large abscess immediately beyond the occluded bronchus. In this case we have an abscess in the opposite lung, not at all connected with the tumor so far as we can make out.

He had in addition a very marked hypertrophy of the heart and a moderate chronic vascular nephritis,—hypertensive heart disease.

DR. FREDERICK T. LORD: The finger-like projection in the bronchus is of interest with respect to the possibility of error in bronchoscopic localization. If bronchoscopy had been done, the appearance might well have suggested that the mass arose from the region of the tracheal bifurcation, whereas it actually took its origin from a much lower level and might conceivably have been treated surgically with success at an earlier period in its development.

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SHALL THERE BE A REDUCTION OF THE NUMBER OF MATRICULANTS IN MEDICAL COLLEGES?

A RESOLUTION adopted by the Fulton County and Cobb County Societies both of Georgia as appears on page 901 of this issue, has been forwarded to medical journals with the expectation of appropriate action by State Medical Societies.

This is prompted by a laudable desire to improve the financial status of the average doctor who finds that there are too many competitors and who is now struggling to support his family and educate his children.

That there is a numerical plethora of doctors is well known, but it is equally recognized that the greater difficulty lies in the uneven distribution of practitioners, for many rural areas are not adequately served.

It is also apparent that a substantial proportion of doctors do not keep abreast of progress in the employment of scientific details of practice and this in turn diverts practice from the average doctor into special fields.

Whether a reduction of the number of graduates in medicine would overcome the well-known financial difficulties of the rank and file in the profession is a question without a satisfactory answer at the present time, because the economic status of the low income groups of the population absolutely prevents the payment of a proportion of the doctor's fees by reason of the diversion of too large a part of the income in this group into channels which might be reduced if less money were spent on automobiles, cosmetics, movies and other entertainments.

The indisposition of the average working man to budget his income will continue to operate to the disadvantage of the doctor.

The medical profession can, with propriety, take some action with respect to adjusting the relation of specialists and family physicians as well as organization designed to prevent the too general use of charitable medical service.

We are of the opinion that when the poorly equipped doctor shall have been eliminated and the cults driven from the field that then will be a very proper time for a study of the reduction of the number of medical students. When that time comes, however, may it not be best to refer this matter to the Association of American Medical Colleges rather than to the American Medical Association? If, however, the American Medical Association will devote study to this subject it may be of definite assistance to the American Association of Medical Colleges.

JOSEPH F. F. BABINSKI, 1857-1932

THE death of Dr. Joseph Francisco Felix Babinski, October 30, in Paris, France, removes from the medical world a figure of international fame. He died at the age of seventy-five, after a career in neurology and medicine that made his name known in every clinic and to almost every doctor in the world. Babinski was one of the founders of the Society of Neurology of Paris, a member of the French Academy of Medicine and a commander of the Legion of Honor.

Of Polish descent, although born in France, November 18, 1857, Babinski graduated in medicine from the University of Paris in 1885 with a thesis on multiple sclerosis[1], of outstanding value for its time. This paper was the first of a long series of articles, the chief of which were gathered together in an extensive, documented bibliography, in 1913[2]. Here one finds extracts from his papers, as well as the striking illustrations so characteristic of Babinski's publications. This work, one of extreme value, gives a clear idea of the active nature of Babinski's mind and the multiplicity of his investigations into practically all of the divisions of

neurology. He added many signs to clinical medicine, the most important being the reflex which goes under his name.

Babinski's sign was first described in a preliminary note read before the Société de Biologie, February 22, 1896; it was a simple statement that, "in certain cases of 'paralysis', the toes on the affected side, instead of flexing when the sole is stimulated, execute an extensor movement on the metatarsal"[3]. Two years later[4], he gave a full account of this phenomenon and his excellent description summarized the important features of the normal and pathological plantar reflex in human beings. To his description there has been little to add, in later years, although the fanning of the outer toes, which often is part of the reflex, was not mentioned in the original paper, but was separately described in 1903[5]. An immense literature has grown up surrounding Babinski's sign since the date of the original description and it is now firmly established that this reflex, indicating disease or injury of the pyramidal pathway, is only second in importance to the Argyll-Robertson pupil as a sign of a structural lesion of the central nervous system. Babinski's description was soon brought to the attention of the English-speaking medical world by the paper of James Collier, published in 1899[6], in which Collier first fully appreciated the functional significance of the normal plantar reflex and its pathological counterpart, the Babinski sign. More recently, this reflex has been the subject of an extensive investigation by Professor John F. Fulton of the Yale Medical School[7]. He has traced the development of the reflex through the primate series of animals in his "The Sign of Babinski; a Study of the Evolution of Cortical Dominance in Primates." In Professor Fulton's monograph one will find a translation of Babinski's original description, a discussion of the subject of the plantar reflex in general, as well as a brief summary of the clinical implications of bilateral cortical lesions as noted in traumatic cerebral diplegia and in Little's disease.

As a clinical neurologist and teacher Babinski held a high place in Paris for many years, although somewhat overshadowed by Pierre Marie and Jules Déjerine. Originally chief of Charcot's clinic, Babinski later developed his own clinic at the Hôpital de la Pitié, one of the more modern hospitals in Paris, situated not far from the famous Salpêtrière. Here his clinic grew to great proportions; a good description of his justly famous Monday morning demonstrations is contained in an article by Dr. Malford W. Thewlis[8].

Babinski's work on functional diseases has been rather obscured by the eponymic character of his description of a single reflex. He added greatly to our knowledge of nervous disorders in general, playing a particularly important rôle in postwar Paris, where he had hundreds of soldiers under his care. By his death medicine loses a great figure, a man who will always be remembered on account of his name's being attached to an important reflex, but whose contributions to the whole subject of neurology were of outstanding value.

REFERENCES

1 Étude anatomique et clinique sur la sclérose en plaques. Thése pour le doctorat en médecine par J. Babinski. Paris, 1885.
2 Exposé des travaux scientifiques du Dr. J. Babinski. Paris: Masson & Cie, 1913.
3 Compt. rend. Soc. de biol. 3:207, 1896.
4 Semaine méd. 18:321, 1898.
5 Rév. neurol. 11:728, 1205, 1903.
6 Brain 22:71, 1899.
7 The sign of Babinski: a study of the evolution of cortical dominance in primates, by John F. Fulton and Allen D. Keller. Springfield, Ill. & Baltimore, Md.: Charles C. Thomas, 1932.
8 New York Med. J. 115:295, 1922.

MASSACHUSETTS HAS SPOKEN

THE petition of the chiropractors of Massachusetts for endorsement of this cult by the appointment of a separate board for the registration of their practitioners, as it appeared on the ballot before the voters of this Commonwealth at the last election, was rejected by a plurality vote of well over one hundred thousand. The result was secured by a united profession which carried on a campaign of instruction reaching to almost all the communities of the state. This shows that organization and enthusiasm is effective when inspired by a purpose to protect the people from definite dangers.

A campaign of this character requires the expenditure of considerable sums of money which were provided by local groups supplementing appropriations by medical societies.

The appeal to the voters to vote No was founded on the advantages of the single standard of education and experience required to meet the examinations of the Board of Registration in Medicine and was not a defensive movement by physicians. The more general understanding of the state requirement will strengthen appeals for state recognition of higher standards for registration of all who may wish to assume the responsibilities of caring for illness.

It may be of interest to note the following recorded facts respecting the votes cast in some communities:

	Yes	No
Alford	10	23
Ashland	260	319
Attleboro	2,152	3,056
Becket	76	95
Beverly	6,073	4,787
Brockton	7,239	8,771
Cambridge	8,178	15,809
Chelsea	2,369	3,423

	Yes	No
Cheshire	140	253
Chicopee	3,545	3,112
Clarksburg	137	112
Dalton	425	877
Dedham	1,332	2,474
Egremont	44	69
Everett	4,653	4,578
Fall River	4,826	16,581
Fitchburg	3,618	5,146
Florida	13	40
Gloucester	2,367	2,452
Great Barrington	503	787
Hancock	52	69
Haverhill	4,107	6,593
Hinsdale	113	78
Lanesboro	125	82
Lawrence	6,854	5,972
Lee	300	573
Lenox	248	447
Leominster	2,066	2,851
Lowell	7,085	14,621
Lynn	8,801	15,116
Malden	5,412	7,267
Marlboro	2,439	1,509
Medford	5,741	9,814
Melrose	2,126	6,698
Monterey	24	57
Mt. Washington	9	14
New Ashford	6	6
New Bedford	7,838	12,933
Newburyport	1,801	1,607
New Marlboro	73	93
Newton	5,367	18,259
North Adams	2,066	2,591
Norwood	1,125	2,205
Otis	25	54
Peabody	1,378	2,454
Peru	12	14
Pittsfield	4,004	7,942
Quincy	7,634	11,729
Revere	2,667	3,633
Richmond	61	118
Rockland	650	1,134
Salem	2,961	6,029
Sandisfield	20	24
Savoy	17	34
Sheffield	147	182
Somerville	9,166	14,336
Springfield	12,431	18,968
Stockbridge	184	309
Taunton	2,695	4,285
Tyringham	24	39
Waltham	4,701	5,101
Washington	19	32
West Stockbridge	105	174
Williamstown	347	705
Windsor	34	65
Woburn	1,898	3,101
Worcester	12,532	30,192
Totals	175,450	292,873

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

IRWIN, RALPH L. M.D. State University of Iowa 1925. Pathologist to Sanatorium Division, Boston City Hospital. His subject is "The Essential Pathology Found in Diphtheria, Scarlet Fever and Tuberculosis." Page 863. Address: Boston City Hospital, Boston.

PLACE, EDWIN H. M.D. Harvard University Medical School 1904. Physician-in-Chief, South Dept., Boston City Hospital. Clinical Professor of Pediatrics, Tufts College Medical School. Consultant, Contagious Diseases, Massachusetts General Hospital, Massachusetts Memorial Hospitals, etc. His subject is "Heart in Diphtheria and Scarlet Fever." Page 864. Address: 745 Mass. Ave., Boston.

HAWES, JOHN B. 2nd. A.B., M.D. Harvard University Medical School 1903. President, Boston Tuberculosis Association. Director, Massachusetts Tuberculosis League and National Tuberculosis Association. His subject is "The Heart in Pulmonary Tuberculosis." Page 874. Address: 11 Marlborough St., Boston.

RILEY, AUGUSTUS. A.B., M.D. Harvard University Medical School 1907. F.A.C.S. Assistant Professor of Genito-Urinary Surgery, Harvard Medical School. Visiting Surgeon in Urology, Boston City Hospital. Urologist, Malden Hospital. Consultant in Genito-Urinary Surgery, Symmes Arlington Hospital. Consultant, Genito-Urinary Department, Boston Dispensary. His subject is "Conservative Kidney Surgery." Page 877. Address: 868 Beacon St., Boston.

THENEBE, C. L. M.D. University of Pennsylvania 1918. Visiting Epidemiologist, Hartford Isolation Hospital. Visiting Pediatrist, St. Francis Hospital and Mt. Sinai Hospital, Hartford. Address: 68 Pratt St., Hartford, Conn. Associated with him is

CENCI, V. P. M.D. Tufts College Medical School 1929. Resident Physician, Hartford Isolation Hospital. Address: Hartford Isolation Hospital, Hartford, Conn. Their subject is "Treatment of Acute Anterior Poliomyelitis with Non-Specific Protein. A Preliminary Report." Page 880.

ALLEY, LEON A. M.D. Tufts College Medical School 1915. Superintendent, Lakeville State Sanatorium. Formerly Assistant Superintendent, Rutland State Sanatorium and Assistant Physician, Boston Sanatorium. His subject is "The Sanatorium Child." Page 884. Address: Lakeville State Sanatorium, Middleboro, Mass.

MISCELLANY

AN EXPRESSION OF APPRECIATION

The Committee on State and National Legislation desires to express its sincere appreciation to the members of the Massachusetts Medical Society and to the members of our allied professions, as well as to the several philanthropic, public health and public welfare organizations, for the interest which they have shown in opposing the chiropractic petition.

The Society is also indebted to the *New England Journal of Medicine* and to the press throughout the State. They have been most liberal in their Editorials.

To the hospitals, to the various Boards of Health, to the Tuberculosis League and to our esteemed lay friends, we can only acknowledge our indebtedness and express our appreciation for the good work accomplished by them.

HALBERT G. STETSON,
THOMAS J. O'BRIEN,
FREDERICK E. JONES,
ARTHUR W. MARSH,
SHIELDS WARREN.

A RESOLUTION*

To the Secretaries of the County Medical Societies:

The following resolution was passed by the Fulton County and Cobb County (Georgia) Medical Societies at their regular meetings a few weeks ago after the reading of a paper entitled "The Economic Status of the Medical Profession," which appeared later in the *Journal of the American Medical Association*, October 15, 1932, Page 1358, Under the Department of Medical Economics.

Since this article appeared, we have had numerous letters from physicians all over the United States urging that this plan be placed before the different county, state and national societies for their consideration and adoption.

We are asking that you place this or a similar resolution before your society and ask for its adoption. Have it signed by your proper officials and forward to the secretary of the Medical Association of your state.

These resolutions will be placed in the hands of the delegates to the American Medical Association at its next annual meeting.

RESOLUTION

Whereas, the medical profession of the United States is suffering losses all out of proportion to its ability to endure, and

Whereas, our losses are not so much due to the financial depression as to over-production of doctors and decreased demand for paid medical service generally, and

Whereas, the production of doctors is far in excess of the population increase,

Therefore, we the members of the ..... County Medical Society respectfully request the Medical Association of ....., the other State Medical Associations, the Southern Medical Association and the American Medical Association to adopt this or similar resolutions requesting the Medical Colleges of the United States to reduce the number of graduates each year until the law of supply and demand has been fully complied with. The number of graduates to be determined by a national committee appointed by the President of the American Medical Association.

Respectfully submitted,
——, M.D., *President.*
——, M.D., *Secretary.*

*Forwarded by B. T. Beasley, M.D. of Atlanta, Georgia.

APPOINTMENTS TO THE FACULTY OF BOSTON UNIVERSITY MEDICAL SCHOOL

The appointment of two professors emeriti, promotion of three faculty members, and the naming of six men to instructorships and eight men to assistantships, was announced recently by Dr. Alexander S. Begg, dean of Boston University's school of medicine, previous to his leaving for Philadelphia to attend the conference of the Association of Medical Colleges.

Dr. Martin J. English, well-known Boston physician, who was formerly associate professor of Pediatrics, has been named full professor and head of the department of Pediatrics, the position which was left vacant by the recent death this fall of Dr. Orville R. Chadwell. Dr. English who was graduated from Harvard Medical School in 1907 is also pediatrician-in-chief at four hospitals including Boston City Hospital, St. Elizabeth's Hospital and St. Margaret's Hospital, Boston, and Whidden Memorial Hospital, Everett. He is a trustee of Boston City Hospital, a member of the Whidden Memorial Hospital medical advisory board, and a member of the American Medical Association, Massachusetts Medical Society, New England Pediatric Society, and American Association of Teachers of the Diseases of Children.

The two faculty members who are named professors emeriti are Dr. Solomon C. Fuller, professor emeritus of Neurology, who came to the school in 1909 in the department of anatomy, and Dr. Conrad Smith, professor emeritus of Laryngology, who joined the staff in 1912.

Herman C. Petterson, M.D., has been named associate professor of Pediatrics, and Moses J. Stone, M.D., assistant professor of Diseases of the Chest. New appointments are as follows:

Instructors: John H. Cauley, M.D., Clinical Medicine; Jacob Kaminsky, M.D., Tuberculosis; James H. Peers, M.D., Pathology; Clifton T. Perkins, M.D., Clinical Psychiatry; Andrew Peters, M.D., Tuberculosis; Morris Yorshis, M.D., Clinical Psychiatry.

Assistants: Welman B. Christie, M.D., Anatomy; Kenneth K. Day, M.D., Anatomy; Theodore K. Keith, M.D., Anatomy and Surgery; Elihu Lewis, M.D., Medicine; Thomas R. Mansfield, M.D., Anatomy; Lee MacPhee, M.D., Gastro-Enterology; Ensio

K. F. Ronka, M.D., Surgery; Henry N. Rosenberg, M.D., Medicine. — *Boston University Medical School Bulletin.*

AID FOR HOSPITALS

That the financial condition of the voluntary charitable hospitals of this country is becoming acute is shown by Dr. William F. Snow, President of the National Health Council in New York City, who has issued a warning to the public against forcing these hospitals to lower their medical standards. He sets forth that these hospitals have served more than 5,000,000 patients during the last year and that their finances are at such a low ebb on the average that contributions by the public must be liberally given if it expects them to continue to give the best that medical science can offer. He pointed out that the spread between income and expense has been greatly increased in the last three years so that on the average they now are giving more than 30% of their services to patients who cannot pay the cost of their care, while their earnings have fallen off from 15 to 20 per cent. The American people have been paying something less than $100,000,000 for the support of this free work.

DR. E. J. CRANE IS ASSOCIATED WITH THE HOLDEN CLINIC

Edward J. Crane, B.S., M.D., of Lawrence, Massachusetts, is becoming associated with the Holden Clinic. Dr. Crane graduated at Tufts College Medical School in 1930. He interned for six months at the Boston City Hospital, Division of Tuberculosis, at Mattapan, and for two years at the Worcester City Hospital.

CORRESPONDENCE

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
October 29, 1932.

Managing Editor,
The *New England Journal of Medicine,*

In addition to the articles enumerated in our letter of September 24 the following have been accepted:

Lederle Laboratories
Solution Liver Extract (Lederle) for Oral Use

Parke, Davis & Co.
Diphtheria Toxin Diluted for Schick Test
Diphtheria Toxoid, 30 cc. vial hospital packages

The following articles have been included with the list of Articles and Brands Accepted by the Council But Not Described in N.N.R. (New and Non-official Remedies, 1932, p. 487):

Smith Oil & Refining Company
Smith's Mineral Oil

Yours very truly,
PAUL NICHOLAS LEECH, *Acting Secretary,*
Council on Pharmacy and Chemistry.

OUR MAJOR VICTORY

Mr. Editor,

Whatever pangs the election may have brought to some of our Fellows, we are all happy in our major victory—the defeat of the Chiropractors. To have won with no covetous motive and no undignified action is all the sweeter.

The Shade of Dr. Holyoke (so vividly brought back to us recently by Dr. Cushing) can now take heart again. In the years to come, as we separately step out to meet him, we can be sure of an earnest welcome. May many more years pass before Dr. Thomas J. O'Brien has that pleasure!

Yours,
DWIGHT O'HARA, M.D.
520 Commonwealth Avenue, Boston.

RECENT DEATHS

CHAPMAN — WILLIAM HARDEN CHAPMAN, M.D., died at his home in Hingham, November 6, 1932, aged 44. He maintained a Boston office at 496 Commonwealth Avenue. He graduated in medicine from the Boston University Medical School in 1919 and joined the Massachusetts Medical Society in 1920. He was a member of the Appalachian Mountain Club, the Harvard Club, and the South Shore Country Club.

A mother, four sisters, and a brother survive him.

SMITH—DR. FRED STEVENS SMITH died in North Andover, Massachusetts, November 2, 1932, where he had practiced nearly forty years.

He was born in 1870 and graduated from the Harvard University Medical School in 1895 and joined the Massachusetts Medical Society in 1885.

Dr. Smith was much interested in civic affairs and was chairman of the school committee at the time of his death.

His widow and two daughters survive him.

WALCOTT — HENRY PICKERING WALCOTT, A.B., LL.D., M.D., died in Cambridge, November 11, 1932. Dr. Walcott was born at Salem, Mass., December 23, 1838, the son of Samuel Baker and Martha (Pickering) Walcott. He graduated from Harvard College in 1858 and from the Bowdoin Medical School in 1861. The desire to enter service in the Civil War led to his taking the M.D. degree from Bow-

doin rather than waiting the required time at the Harvard Medical School where he had studied for nearly three years.

After his retirement from the Army he resumed the study of medicine in Berlin and Vienna. On returning to the United States he entered practice in Cambridge, and soon after began a long public career, first drafted to reorganize the administration of the Tewksbury State Almshouse. Soon after this he accepted the position of chairman of the State Board of Health, continuing in this office until 1916. He was to a large degree the controlling mind in the creation of the present Metropolitan Water System and served as its chairman for many years.

In many other ways Dr. Walcott served the state in initiating and developing public health activities. Although drafted into many fields of responsibility such as overseer and later Fellow of Harvard University, Trustee and for a time President of the Massachusetts General Hospital, he never lost interest in problems of medicine and the Massachusetts Medical Society, serving the last-named institution as its president, 1896-1898.

His influence extended throughout and beyond the United States as shown by memberships in many scientific bodies.

Although a sufferer from physical infirmities for many years, his mind was active and his interest in current affairs keen up to a few days before his death. His personal friends found enjoyment and inspiration in frequent visits to the gifted physician and public servant.

Dr. Walcott is survived by a son Judge Robert Walcott, Associate Justice of the District Court at Cambridge.

An appropriate obituary will be published at a later date.

NOTICES

REMOVAL

HERMON NORTON, M.D., announces the removal of his office to 209 Pleasant Street, Newton Centre, Mass.

RADIO HEALTH MESSAGES

NOVEMBER-DECEMBER, 1932

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZ. Tuesdays, 4:15 P.M.

November

22 **Only a Sore Throat**
29 **Anemia or the Lack of Blood**

December

6 **Sickness and Character**
13 **How Are You?**
20 **Chorea or St. Vitus' Dance**
27 **Hemorrhoids**

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays, 12:30 P.M.

Health Subjects of Public Interest discussed by the Division Directors of the Department.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 4:50 P.M.

Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.

REPORTS AND NOTICES OF MEETINGS

A WELCOME TO DR. ELLIOTT C. CUTLER

On the evening of November 7 a large group of the friends and associates of Dr. Elliott C. Cutler gathered in the Aesculapian Room of the Harvard Club to express their pleasure in his return to Boston to take the position of Moseley Professor of Surgery at the Harvard Medical School and Surgeon-in-Chief, Peter Bent Brigham Hospital.

At the conclusion of the banquet, Dr. Henry A. Christian called the meeting to order and, after tendering to Dr. Cutler the cordial welcome of the company, called upon Dr. Harvey Cushing to speak more at length. Dr. Cushing built his address on the record of the one hundred and fifty years of surgical teaching at the Harvard Medical School, humorously explaining to Dr. Cutler that this occasion establishes a precedent in that the appointment of his predecessors in no instance was celebrated by a meeting of this character.

Following a reference to the action leading to the invitation to Dr. Cutler to come to Boston, Dr. Cushing referred to each of Dr. Cutler's predecessors who occupied the chair of Professor of Surgery at Harvard explaining that the title conferred upon Dr. John Warren, the first of the series, included that of Professor of Anatomy. In speaking of these men in turn he cited personal characteristics and the years of service of each. Following John Warren there was John Collins Warren, George Hayward, Henry Jacob Bigelow, David W. Cheever, J. C. Warren, Maurice W. Richardson the speaker and now the present incumbent.

An interesting feature of the services of these leaders in surgery was the long period of occupancy of this chair by several of the group who were not subject to conditions of retirement now in force.

The address was replete with historical facts and made especially interesting by witty allusions to personal characteristics of individuals and correlated circumstances.

In responding, Dr. Cutler referred to the delightful associations of his earlier work in Boston and his conviction that it is often well for a professional person to broaden his experience by going away from

early environment into new fields and meeting the problems of adjustment to other conditions. He felt that his return to Boston after nine years in the middle west would provide opportunities for further advance in his chosen field.

He explained his reactions to the questions which were uppermost in his mind when planning his career as to whether it were better to develop personal service of the clinical surgeon or that of the investigator who might more definitely restrict his time and energies to the elucidation of those mysteries which scientific medicine is trying to solve. Even with these two attractive special fields open to ambitious minds, he felt that organization of a teaching department in a medical school which prepares its students for the application of scientific surgery to the ills of people is especially appealing and worthy of one's best efforts.

REPORT OF THE EIGHTH INTERNATIONAL CONFERENCE AGAINST TUBERCULOSIS

(Continued from Page 861, issue of November 10)

POST-SANATORIUM ASSISTANCE

Dr. B. H. Vos, Superintendent of the Public Sanatorium of Hollendoorn (Netherlands) made the opening report in French. An English translation was printed. He reviewed the early days of the subject and showed the differences between "Occupational Therapy" and "Sheltered Employment."

There are more and different types of cases needing sheltered employment now than in 1922 because of improved methods of treatment. Apart from the many who leave the sanatorium ready to resume active work and those who need temporary sheltered employment, there are many who can never be assigned to permanent work without the risk of relapse and who would need immediate rest treatment.

Dr. Vos told of efforts at providing sanatoria with facilities, etc., industrial shops and settlements in various countries. Conditions are so various that no one type of organization can be the rule.

Agricultural Settlements seem to suit better in France than elsewhere. They are not so useful in Holland or England. Agricultural work is usually alternately light and heavy.

The Dispensary should be the controlling factor in examining and approving plans as to what a patient should do and where he might go.

No pay should be given for work so long as it is of therapeutic value, but it must be suitably remunerated as soon as the patient is making use, so far as possible, of his reduced working capacity.

To provide for the patient and his family, some form of subsidy will be necessary either temporarily or permanently. Village settlements and workshops are two of the schemes for helping patients. In addition there will be needed a vocational service centre where each case can be considered on its own merits. A centre of this kind was established in New York in 1923. In both settlement and industrial shop there must be close coöperation between the physician and the technical director. There is need of a union of local committees to help in the financing and coöperative management of all settlements and industrial shops.

Constant medical supervision is indispensable for the efficiency of after-care schemes.

There were ten co-reporters whose abstracts cover fifty printed pages, followed by five-minute discussion papers by about twenty-five others.

Dr. J. Blanco, "Tablada" Sanatorium, Madrid, gave the first co-report.

The dispensary should be the first of the system. It recommends the patient to the sanatorium. When the patient is ready to leave the sanatorium he should go to work in one of the suburban shops created by independent means but under the direction and control of the dispensary. The shops could make favorable contracts to supply articles needed by public institutions and hospitals. The dispensary could classify the patients who would be assigned by a placement office. The patients would continue to occupy their own homes or if necessary inexpensive quarters not too far from the shops. In case of a relapse the dispensary doctor should decide to what hospital or sanatorium the patient should go.

It is clear that patients having little chance of regaining the power to work should remain in a hospital.

The problem could be solved by creating in all hospitals and sanatoria a group of "Invalids at Work," arranging for special shops and inexpensive near-by living quarters.

Dr. Edouard Bresky of Prague reported for the tuberculosis medical specialists of Czechoslovakia.

The after-care or assistance depends upon an efficient sanatorium treatment. It also depends on the proper selection of cases. About 30 per cent of the tuberculous patients are sent by their physicians too late to get the more favorable treatment and regain the capacity for work.

The basis of all treatment is a well-functioning dispensary, not only treating the individual but interested in the needs of his family.

Patients discharged from sanatoria can be classified in four groups as follows:

1. Invalids capable of work who need permanent oversight.
2. Invalids needing supplementary treatment before being assimilated into group 1.
3. Invalids needing to be reëducated in another profession before becoming capable of being employed.
4. Invalids incapable of work for whom there is little hope of recovering capacity for being employed.

Dr. Ernst Brieger, medical chief of the Municipal Hospital for Tuberculosis at Breslau, describes the colony that has grown up in close association with his hospital. He thinks the neighborhood of a hospital is better for a colony than that of a sanatorium. Important considerations oppose the introduction of

a work cure of short duration following rest treatment.

The description of his unique colony deserves attention. Besides lodgings for fifty men and for fifty women there are thirty-two single family pavilions. It is not a village settlement like Papworth for the married patients are still ill, under medical supervision, and can, by working a four-hour period each day at one-half standard wages, earn a supplement to their rent allowance.

Various industries are in operation specializing in articles needed by hospitals and public institutions which take half their product. They are furnished stock and have no capital and thus depend for a market on those interested in the problems of tuberculosis.

When ex-patients can work from six to seven hours a day they may continue in the colony and may become instructors or foremen. This colony has grown to meet a need without any preconceived plan. It was planned to diminish rather than increase the cost of assistance, gradually to free the patient from the paralyzing atmosphere of the hospital and to stabilize the clinical recovery. It is following the method of organic evolution of sanatorium care without losing sight of other methods of post-sanatorium assistance.

René Courtois and Edgar Olbrechts reported for Belgium. The former has charge of a sanatorium for men at Marcinelle, where the work plan was begun three years ago. This has been a very satisfactory experiment. Two other experiments are under way.

Those capable of full work or only part-time work and those unable to do any serious work comprise the groups of sanatorium graduates.

During 1929, 155 patients were discharged. 1. Forty-six have 100% capacity and have found work. 2. Nineteen have recovered a partial work capacity. 3. Thirty-three have no work capacity. 4. Thirty-one died during the first year. 5. Twenty-six were not tuberculous.

During 1930 and 1931, 291 patients were discharged. 1. Seventy-seven have 100% capacity. 2. Forty-two have partial work capacity. 3. Sixty-seven have no work capacity. 4. Sixty-one have died during the first year. 5. Forty-four were not tuberculous.

About 33 per cent had complete work capacity. About 16 per cent had partial work capacity. Hours of work vary from 2 to 8. Period of reëducation often passes ten weeks. The work is not paid. The sole privilege of the workers is a few days' leave. The choice of an occupation is a matter for individual study.

Dr. Louis Guinard of Bligny mentioned several sanatoria in France that have patients at various sorts of work. He told of a projected village at Salagnac (Dordogne) that will be ready in the spring of 1933 for 3000 inhabitants. Each cured patient will enjoy an annual pension of 20,000 francs!

An inquiry of former patients at Bligny gave the number of men and women who were able to resume work, 1422 altogether. A few had relapses but no more among those who resumed their former occupations than among other groups. It is clear that those whom the "cure" has benefited are those who, independent of their occupation, have lived in the country under the best hygienic conditions. More successful also were those who could prolong their rest and resume their occupations progressively.

Out of an average of 545 pensioners at Bligny there are from 275 to 280 who have an artificial pneumothorax; these will need assistance.

Dr. Guinard gave ten general conclusions of which the sixth is as follows: The public fear of tuberculosis has now turned not only against the patients but against the best means employed in the social and medical defense against the disease, so that one ought now to counteract this current, that larger and better proposals for "after-care" may be more readily accepted.

Dr. Nils Heitmann, of Oslo, reported for Norway. Since 1911 the state has made an annual appropriation for aiding patients leaving sanatoria or homes for the tuberculous. The money is paid to towns or local committees that must raise an equal sum.

Contagious patients are isolated in houses of from 16 to 50 beds of which there are 120 scattered about the country.

In 1925 there was built in Eastern Norway a large sanatorium. Near this a small private sanatorium was bought and turned into a work place for 30 men patients. Some had no desire to work and some were eager for it. They knew they were under the supervision of a medical specialist. The patients agreed to stay a year. Their rate of pay was increased every three months. From two hours a day they gradually increased to six. No more was allowed. At least six trades were taught of which the patient chooses one. The shop sells its products to institutions and other clients but has severe competition from prisons and other state shops. Patients seldom have relapses and usually regain their weight and optimism. Other shops have been started and still further progress will be made when times are better.

Prof. Eugenio Morelli of Rome reported for Italy. He has had supervision of the construction of 20,000 beds in sanatoria all over Italy and 3000 beds in post-sanatorium colonies. By the end of this year 1932, 12,000 beds in sanatoria will be available and three large colonies will be well under way.

In Italy the problem of the colonies is a national one closely associated with the sanatorium problem on which it depends.

He discusses in detail the construction and use of colonies including those for "improved patients".

HIS CONCLUSIONS

1. It is necessary that the sanatoria be closely related to the colonies, from the sanitary as well from the economic point of view.

2. The products of the colonies should be used by the sanatoria.

3. The colonies should include agriculture and other industries.

4. It is necessary to establish colonies even for "improved patients".

5. This organic and harmonious undertaking is possible only if all the antituberculous therapeutic activity is spread by the large institutions or better still by the nation itself, as we do in Italy.

The Italian people bless their grand Chief, to whose good will they owe this insurance law.

Dr. H. A. Pattison of Livingston, New York, presented a report from the American point of view. He defined more clearly the term "occupational therapy" as distinguished from "vocational therapy," "ergotherapy," and "industrial convalescence". He spoke of institutions where the patient could immediately start working if he had reached the quiescent stage of the disease. He referred to several institutions in the United States, accounts of which have been published by the National Tuberculosis Association and elsewhere.

An Advisory Committee has suggested more rigid criteria for determining the eligibility of candidates for ergotherapy. One of the most troublesome problems is to devise standards for ability to work. One can only test the individual patient, though x-ray examination and leucocytic counts may help.

Many cases needing after-care treatment contracted tuberculosis while in school and, therefore, have no trade. It is the more difficult to return them to society because of their inability to find suitable work at an adequate wage.

The selection of industries to be conducted in after-care institutions is exceedingly difficult, especially those suitable for women. Several patients have been found to do well and gain weight while employed as night watchmen. Dr. Pattison finds that the duration of rest treatment and of rehabilitation work cure must be considerably longer than the intervals mentioned by Dr. Vos, but agrees with him on all other points.

Dr. David A. Stewart, Superintendent, Sanatorium Board, Manitoba, Canada, was unable, on account of ill health, to be present. His printed report under the title of "The Sanatorium, a School" deserves to be widely read. Half an hour a week per patient of teacher-time would convert the sanatorium into a school where interest could be developed in cultural, avocational, and even vocational studies of great value to every individual patient and the result would also be of great value to the institution. "For the excellency of knowledge is, that wisdom gives life to them that have it."

Dr. Harley Williams, Secretary of the National Association for the Prevention of Tuberculosis of Great Britain, presented a report covering the details of "Care" and "After-care" of tuberculous individuals.

1. Schemes of social supervision which regard the tuberculous family as a unit in its own home, and as a rule, conducted by the tuberculosis dispensaries.

2. Occupational Therapy in Sanatoria, where the idea of training in a particular trade is subordinate to the question of treatment.

3. Vocational Training in Colonies.

4. Industrial Settlements.

"Britain, in common with the whole world, is passing through an unprecedented period of economic difficulty, and it is no criticism of schemes for care of the tuberculous that they fail, when we know that the normal social organization for healthy persons also fails conspicuously. Tuberculosis has a sinister reputation for its pauperizing powers, and these must be countered with ways to meet the needs of the individual patient. While we may search for the ideal method to be employed universally, we should not neglect any system which may help in a case under review. The ultimate test of all such schemes is whether they regard the tuberculous family as a unit, and pay adequate consideration to continuity of care and the preventive measures calculated to protect the children."

A number of five-minute papers were presented and then Dr. Vos closed the discussion naming his points of agreement or disagreement with several of the speakers.

CALVIN G. PAGE, M.D.

COMBINED MEETING OF THE PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE, PHILADELPHIA PEDIATRIC SOCIETY, AND THE NEW ENGLAND PEDIATRIC SOCIETY

The combined meeting of the Pediatric Section of the New York Academy of Medicine, the Philadelphia Pediatric Society, and the New England Pediatric Society was held in one of the amphitheaters of the Harvard Medical School on Saturday, October 15. Dr. Lewis Webb Hill, President of the New England Society, presided. The gathering heard eleven papers at its two sessions.

Dr. W. B. Cannon delivered the first address at the morning meeting, choosing as his subject "An Interpretation of the Functions of the Sympathetic Nervous System." Dr. Cannon first briefly reviewed the anatomy of the autonomic nervous system, pointing out that the cranial and sacral divisions send long preganglionic fibers to ganglia located in or near special organs, whereas, the same fibers of the sympathetic system (thoraco-lumbar or mid-division of the autonomic) proceed to a chain of ganglia located near the spinal cord. The sympathetic system is thus arranged for diffuse activity, the cranial autonomic is a protective (closes iris, slows the heart, etc.), conservative, and upbuilding unit, and the sacral autonomic is concerned with the emptying reflexes. The sympathetic system also innervates the adrenal medulla, which interesting organ produces the substance adrenin, which acts similarly to the sympathetic itself.

The use of the denervated heart in experimental studies with adrenin was next described. Animals with denervated hearts react to cold with an in-

crease in the pulse rate, but if the adrenals be removed, the rate does not rise—in fact it goes down a little. On the other hand, animals in such a situation, and with the adrenals removed rely more on shivering in an attempt to maintain body temperature. The adrenal gland thus protects the body against a lowering of its temperature. After excitement, too, if the adrenal is present (still with the denervated heart) the heart rate rises; if it is absent there is no change.

The speaker next discussed the effects of removal of the mid-division (sympathetic) of the autonomic nervous system. There is no effect on the actual *existence* of the animal, growth and development taking place normally. Reproduction in the female is quite possible but there is a disturbance of lactation so that it is hard to rear the offspring. In the male the ejaculation of semen does not take place after the removal of the sympathetic. The reaction of an animal without a sympathetic system to *stresses* is modified. By causing the hairs to stand upon end and by producing surface vasoconstriction the sympathetic system prevents the loss of body temperature. The temperature falls, therefore, when the sympathetic is not present, though it is interesting to find that after a time the animals so treated learn to shiver more effectively and thus compensate in some degree for the change. In hypoglycemia the rate of the denervated heart goes up but if the sympathetic is removed there is no such protection, shock and convulsions soon ensuing. In the struggle the sweating and ridding of the body of excess heat, the rise in heart rate and blood pressure, the contraction of the spleen, and the liberation of some of the sugar reserve in the liver all represent the results of sympathetic activity.

In conclusion, Dr. Cannon discussed the interpretation of the experimental facts. He discussed the interrelationship of man's external and internal environments and noted that the internal environment is kept uniform at all times, chiefly by the action of the sympathetic system as a whole. Occasionally, in pathological states little understood as yet (Raynaud's disease, emotional dyspepsia), the system acts only in part.

The next paper was on "Some Aspects of Secondary Anemia," by George R. Minot. The speaker commenced by discussing five main points under this heading. Mechanical factors interfering with the proper assimilation of food (bad teeth, excessive diarrhea for example), achlorhydria (the presence of acid is probably a factor in the absorption of iron from the upper intestinal tract), an inability of the gastro-intestinal tract to elaborate factors necessary for normal blood formation, improper utilization of blood-forming material after the substances get into the blood, and loss of blood-building material as in chronic blood loss and pregnancy, were each mentioned as having a rôle in the production of an anemia. The degree of deficiency, the speaker went on to say, depends on all of the above factors and successful treatment means replacing the factors on a *quantitative* basis. Adequate therapy does not mean a specific dose of a given drug—the whole procedure varies with the individual. In following a given case the lowest satisfactory gain in hemoglobin is one per cent per day, the average among many of Dr. Minot's patients, two per cent per day. Although copper has proved of value as an adjunct in the treatment of anemia in children, Dr. Minot sees no evidence that it succeeds in adults in the same way.

Discussing the efficacy of various modes of treatment, the speaker presented charts showing that there is no response (the criteria are the presence of reticulocytes and a two per cent hemoglobin rise daily) to Blaud's pills as such, even though the iron be adequate. Powdered pills were fairly satisfactory. Ferric sulphate and sodium bicarbonate, essential constituents of Blaud's pills that are not fresh, were not nearly so effective as *ferrous* sulphate and sodium bicarbonate. Iron and ammonium citrate is by far the best substance to use. It is Dr. Minot's opinion that iron should be given parenterally only rarely if at all, and then in doses not exceeding 16 to 32 milligrams. Alarming symptoms, similar to those of shock, sometimes supervene when doses of over 40 milligrams are given.

Speaking on the subject "Basal Metabolism Determinations in Children: Their Clinical Significance," Dr. Fritz Talbot mentioned difficulties encountered in such studies in children. Necessary for proper interpretation of the results are a general knowledge of the methods, technique, and sources of error, and a knowledge of how the standards are constructed. The growth of the child and the physiologic changes coming with it also must receive due consideration. Though there are many individual exceptions, Dr. Talbot believes that the age of eight years is the lowest limit for obtaining basal metabolic rates satisfactorily with the portable apparatus. Determinations should be made at least 16 hours after food under conditions of absolute rest and with a normal temperature, pulse, and respiration. Results must check within 5 per cent in two or preferably four, periods, and repeated on another day to check again. Discussing the factors of age, sex, sleep, food, and puberty—in the last there is a definite rise of the basal rate, sometimes as much as 35 per cent—the speaker said that if all these preliminary things are borne in mind, the metabolism may then be interpreted correctly. All standards of metabolism are good in the consideration of the average normal child but in the tall-short group with average weight for the height we are limited to the standards of calories per square meter, total calories for the height, or total calories for the weight; in the underweight group total calories for the height, or calories for the expected weight are all right; the only accurate standard is that found in the total calories for the weight of the obesity group. The linear formula Dr. Talbot holds best for the determination of body

surface. In conclusion the speaker added that there is probably only a small percentage of hypothyroidism in conditions like Froelich's syndrome, ichthyosis, eczema, and mongolism, though thyroid in small amounts seems to improve the last-named condition in appearance at least. There is a definite group among adolescent girls where the metabolism may be —18 to —20 per cent and these children are often improved by temporary adequate doses of thyroid. In children, *hyperthyroidism* is specially to be watched for at puberty.

Attempting to answer the question "What Is Celiac Disease?" Dr. John L. Morse presented the next paper on the program. From the point of view of stool examinations there seem to be three types of this disease (1) the type with large, clay-colored, fatty stools, (2) the type with large, loose, yellow-brown, acid and very foul stools, and (3) a combination type resembling each of these other two. Dr. Morse next reviewed the theories of the etiology of the disease. The liver, the pancreas, and the intestine have all been implicated in the origin of the disease. Some have held it to be a disorder of fat metabolism—an excess of secretion, or defective absorption. Errors in mineral metabolism, avitaminosis, and endocrine function have also been held by some to be the cause. All of the theories have fallen down when it comes to proof, however, and the speaker concluded with a clinical interpretation which must serve until the true pathological explanation is forthcoming. The disease is probably not a specific entity but a complex developing late in disease, and progressing. The stools show excess of fats, starch, and fermentation. When the food element doing the harm is eliminated, the condition improves. In these cases of intolerance, there is often a history of overfeeding of the particular elements involved. The prevention of the serious "end forms" of the disease consists in (1) feeding children as individuals, (2) examining the stools when there is an indication of any gastro-intestinal upset, and (3) cutting down on the foods not taken care of in full.

Dr. Henry Christian was the last speaker at the morning session. His topic was "What Is Nephrosis?" The speaker believes "nephrosis" to be a word historically and etymologically ill-adapted to its supposed and apparent meaning. The condition is not a disease or a definite pathological lesion and the term is but a single word, expressing, for the sake of convenience a syndrome, as fully described by Leiter recently in *Medicine*. The word was introduced by Mueller in 1905 and was meant to connote the idea of degeneration as opposed to inflammation (-itis). The true meaning of the word etymologically is "full of kidney substance" and even "-itis" does not literally mean inflammation, but rather that the word used with it is an adjective—thus nephritis would mean "of the kidney". The two outstanding features of the syndrome are persistent albuminuria and edema. Dr. Christian believes that all the evidence (especially the study of certain fish that have no glomeruli and comparing them with others that have) suggests that the cause of the albuminuria is an abnormality of the glomerular membrane.

From the clinical point of view we see three types of patients with the nephrosis syndrome. The first type is the usual patient observed and this patient usually differs from the Leiter definition in a few points—red blood cells in the urine, or high blood pressure, etc. Postmortem observations then show a subacute or chronic glomerular nephritis. Then there is the type where, on first observation, there are no deviations from the Leiter definition. Follow up studies later show deviations as in the first type, and then when the patients die they have typical glomerular nephritis. The third type must be very rare, Dr. Christian thinks, and in this type no glomerular lesions may be found—this might be the very unusual "nephrosis". In other words, in adults at least, the so-called nephrosis is but a stage in the development of glomerular nephritis and it is improbable that there exists a nephrosis in children but not in adults.

Dr. Kenneth D. Blackfan opened the afternoon session with an analysis of the records of 15 cases of vitamin A deficiency observed in children. He reviewed the history and experimental work done on vitamin A deficiency and pointed out that the classical xerophthalmia (with night blindness and characteristic "Bitot spots" on the sclera) is not a full description of the disease. The changes are rather general and systemic, with a keratomalacia wherever epithelium occurs, and not in the eye alone. There has been little mention of this general condition in the literature. The speaker also wished to emphasize the great variety in the symptoms and degree of involvement of various organs and the inconstancy of the sites affected. There is a general stoppage of growth furthermore. A critical examination of the 15 cases has convinced Dr. Blackfan that infection *may* interfere with absorption of vitamin A, but it must not be thought that the vitamin is an anti-infective agent necessarily. The diagnosis of this particular vitamin deficiency is made by a consideration of the history (diet, weight, infection—especially important), the examination of the eyes and skin, x-ray studies (growth), and the presence of keratinized cells in secretions. In closing, the speaker emphasized the fact that this deficiency disease may occur without eye signs (which are late in appearing) and in the presence of what appears to be an adequate vitamin A intake, the condition in the latter instance being due probably to factors inhibiting *fat absorption*, infection, congenital disease, etc.

Dr. S. Burt Wolbach next discussed the pathology of vitamin A lack. The essential features of this are first, an atrophy of the epithelium, and secondly, repair by the basal cells below, with the formation of an excess number of keratinizing cells. Dr. Wolbach demonstrated by means of slides the cessation of growth in bone, the atrophy of the enamel form-

ing organ (epithelial in origin), and the epithelial changes undergone in eyes, lungs, and uteri. The lack of vitamin A predisposes to infection only by the removal of the protective mechanical barrier, and in no specific way. Restoration of normality in animals is started by the seventh day after adequate treatment, and is practically complete by the eighteenth day.

Dr. Gerald Hoeffel conducted a demonstration of patients with endocrine dysfunction. One case of thyrotoxicosis was presented, but particular stress was laid upon hypothyroidism. Cretins are of two types—obvious and masked. To make a diagnosis is of utmost importance if the pediatrician wishes to get a child that will be normal physically and mentally. Early recognition of the condition is aided by x-ray studies, which show osseous retardation, especially in the carpus. In addition to the early recognition of the disease two other equally important factors from the clinician's standpoint are adequate dosage and a persistence in treatment. After a presentation of various types and degrees of cretinism, the pituitary disorders were touched upon. The speaker made the point that the obesity of the so-called Froelich's syndrome can often be controlled and even "cured" by effective dietary measures.

Discussing the "Relation of Anemia of the Newborn to Familial Icterus Gravis and Erythroblastosis" Dr. L. K. Diamond summarized the characteristics of four clinical entities heretofore considered as separate and distinct—universal edema of the fetus, icterus gravis neonatorum or familial icterus neonatorum, erythroblastosis fetalis, and anemia of the newborn. In the first-named condition, first described in 1898, we find generalized edema, enlarged liver and spleen, anemia as seen by the tissue pallor, enlargement and edema of the placenta, extensive extramedullary hematopoiesis, icterus (often marked), and a tendency for the disease to be familial; in the second we find early and severe jaundice, a striking familial incidence, enlarged liver and spleen, abnormally large numbers of nucleated erythrocytes, extensive extramedullary hematopoiesis, dark yellow colored amniotic fluid and vernix caseosa, anemia, and enlargement of the placenta; in erythroblastosis fetalis the spleen and liver are enlarged, there is an extraordinary amount of abnormal hematopoiesis, there is generalized edema of the fetus and placenta, jaundice and pallor of the body tissues are frequent, and there are large numbers of circulating red cells. In anemia of the newborn practically the same signs exist. After a study of several families in which all these conditions are described, Dr. Diamond is convinced that they are all closely related to each other and perhaps stages in the same disease, which is essentially and primarily a disturbance of blood formation or blood destruction. Treatment is symptomatic, numbers of transfusions being indicated primarily. The prognosis is most favorable in the "congenital anemia of the newborn" type.

Dr. Lyman Richards next discussed, with the aid of moving pictures showing a typical case, streptococcus tracheobronchitis in children. The disease occurs most frequently in children aged 2 to 5. It should always be thought of when foreign body, spasmodic croup, and diphtheria have been ruled out, and is sometimes very difficult to differentiate from the last. If diphtheria antitoxin does not cause fairly marked improvement, streptococcus infection should be suspected. The diagnosis is made by laryngoscopy and bronchoscopy, the characteristic findings being a dry, crusted, tenacious secretion on the vocal cord and over the larynx and trachea. The treatment is intubation and, if this fails, tracheotomy. The course of the disease is one of alternating periods of distress and relief. During the times of acute distress immediate bronchoscopy and removal of the crusts are necessary. The pathological picture is one of peribronchial infiltration, the lungs usually being entirely clear.

The last paper of the afternoon session was on "Considerations on the Use of Proteins and Salts in Bright's Disease," by Dr. Allan Butler. Discussing four types of Bright's disease—initial hemorrhagic nephritis, terminal hemorrhagic nephritis, lipoid nephrosis, and latent chronic active nephritis—Dr. Butler gave his views as to the essentials of treatment. In the first type it is essential merely that the calories be kept up and the fluid intake regulated according to the presence of dehydration or edema. Lost blood should be replaced. Magnesium sulphate is indicated when cerebral symptoms appear. In general, the indications for terminal hemorrhagic nephritis are a protein sparing, liberal salt, high caloric diet; and for nephrosis a high protein, salt sparing diet with adequate caloric intake. Transfusion may be indicated to replace lost protein. Guarding against infection is very important. In the fourth type discussed the treatment is variable and must depend on the stage being observed and the character of the blood and urine chemistry.

In the evening, at a dinner at the Hotel Kenmore, Dr. George H. Bigelow spoke on the relationship between public health and pediatrics.

CAMBRIDGE TUBERCULOSIS AND HEALTH ASSOCIATION

At a recent meeting of the Cambridge Tuberculosis and Health Association the following officers were elected to serve for the ensuing year: Dr. Hilbert F. Day, President; Mrs. Rufus P. Williams and Dr. Felix McGirr, Vice Presidents; Miss Louise W. Jackson, Secretary, and William D. Swan, Jr., Treasurer.

MASSACHUSETTS PSYCHIATRIC SOCIETY

At the annual meeting of the Massachusetts Psychiatric Society, held October 28, 1932, at the University Club, the following officers were elected for the following year:

Morgan B. Hodskins, M.D., Palmer, Mass., President.

Winfred Overholser, M.D., Boston, Mass., Vice-President.

Oscar J. Raeder, M.D., 270 Commonwealth Avenue, Boston, Secretary-Treasurer; and A. Warren Stearns, M.D., Billerica, and Harlan L. Paine, M.D., Grafton, Councillors.

The members present were addressed informally by Judge Fosdick of the Superior Court in Springfield on the topic "A Judge Looks at Psychiatry."

WINFRED OVERHOLSER, M.D., *Secretary.*

NORFOLK DISTRICT MEDICAL SOCIETY

The October meeting of the Norfolk District Medical Society was held on Tuesday evening, October 25, in the Empire Room of the Hotel Kenmore at eight o'clock. Dr. S. F. Curran presided. The chief business of the evening consisted in the discussion and acceptance of the amendments to sections X and XIII of the By-Laws of the Society.

Dr. Benjamin Parvey presented a paper entitled "Recent Advances in the Physiology of Reproduction: Zondek-Aschheim Test for Early Detection of Pregnancy." Dr. Parvey pointed out that the hormones found in the urine of pregnant women could also be found in the feces, blood, serum from blisters, saliva, and other body fluids of the same women. The proportion of ovarian and pituitary hormones present varies with the stage of pregnancy. The occurrence of the hormones in the urine of women with hydatidiform moles and tumors of the internal genitalia and in children under ten years of age was discussed. The bilateral hemianopsia and other cerebral symptoms found occasionally during pregnancy were cited as further evidence of hypophyseal enlargement and stimulation.

Dr. Parvey described in some detail the Zondek-Aschheim test in which four immature mice, weighing six to eight grams, are given a series of injections of the unknown urine and autopsied 100 hours after the first injection. A positive test is one in which the following are observed: (1) estrous changes in the vagina and uterus, (2) pin-point hemorrhages in the ovaries, and (3) corpora lutea. The latter two findings constitute positive evidence.

The advantages of Friedman's modification of the Zondek-Aschheim test were discussed, especially the possibility for earlier diagnosis (48 hours after the first injection) and the greater ease of examination made possible by the larger size of the rabbit ovary. Four common sources of error were described: (1) perfume or antiseptic in the specimen bottles destroying the hormones, (2) too great dilution of the hormones suggesting the possibility of a variation in the renal threshold for the individual hormones, (3) unreliability of rabbits under four months of age, and (4) death of the test animals before the completion of the test, often due to contamination of the urine from an unclean bottle. In a series of over six hundred cases, Dr. Parvey reported an accuracy of 99.5%.

The talk was followed by the presentation of a series of lantern slides in which the cyclic changes in the uterine mucosa, and the development of the graafian follicles and corpora lutea were demonstrated.

After a brief discussion of the Chiropractic Referendum by Dr. Thomas J. O'Brien, the meeting was adjourned at 10 P. M.

THE HARVARD MEDICAL SOCIETY

The first meeting of the year of the Harvard Medical Society was held at the Peter Bent Brigham amphitheatre on Tuesday evening, October 25, at 8:15. Dr. Elliott C. Cutler presided. The following cases were presented:

A 60 year old salesman entered the hospital with swollen, painful, red joints. The history of joint trouble dates back 25 years when he first had involvement of his left, great toe. From this the affection spread to other joints. Sodium biurate crystals have been recovered from the joints, and x-ray films show the typical cystic, punched-out areas in the phalanges of the toes and fingers. The blood uric acid content is raised. It was remarked that this is the first typical case of gout in the hospital records since the advent of prohibition.

The second case was that of a 48 year old man with pain in the right shoulder. Ten months previously he had sustained an injury to this shoulder and had kept it in a sling for eight weeks. Motion continued to be painful. He was admitted to the hospital on September 12 for treatment of jaundice, and was advised upon discharge to return for treatment of his shoulder. Examination revealed an atrophy of the supra- and infra-spinati muscles, and marked limitation of all motions in the shoulder joint, particularly abduction. X-ray shows a calcified area in the region of the greater tuberosity. The question arose as to whether this was due to tearing off of the tuberosity, rupture of the supraspinatus tendon or subdeltoid bursitis. It was thought that surgery would be of little value in the former condition and probably of benefit if it were bursitis.

Dr. R. B. Osgood and Dr. P. D. Wilson discussed "Reconstruction Surgery of Chronic Arthritis." Dr. Osgood stated that from the economic consideration arthritis is the most important chronic disease in Europe and America. There are two main forms of arthritis, namely, the atrophic or ankylosing, and the hypertrophic or non-ankylosing types. Surgery is especially valuable in the former.

In order that surgery may be successful, certain criteria must be fulfilled. 1. A subsidence of the active arthritic processes. 2. The patient must be a good operative risk. 3. The patient should be built up in his general health for the operation. 4. Meticulous surgical technique is essential. 5. Economic considerations are quite essential in order that the patient may receive the required postoperative care and physiotherapy to restore motion in the affected member.

The various operative procedures were discussed

by Dr. Osgood and illustrated by slides shown by Dr. Wilson.

In closing, the speakers emphasized that the patients with deformities and crippled limbs are especially appreciative of any slight addendum to the function of their members and that any efforts are worth while in attempting to restore them to usefulness.

THE NEW ENGLAND HOSPITAL ASSOCIATION

The New England Hospital Association will hold its Eleventh Annual Meeting on February 17 and 18, 1933, in Boston.

A. G. ENGELBACH, M.D., *Secretary-Treasurer.*

BOSTON MEDICAL HISTORY CLUB

BOSTON MEDICAL LIBRARY

Monday, November 21, 1932, at 8 P. M.

In Memoriam—Dr. Edward W. Taylor. Charles F. Painter, M.D.

The Influence of Paracelsus on Modern Therapy. E. Melville Quinby, M.D.

"Darwin, Down and Dawn." Benjamin Spector, M.D.

Illustrated by Stereopticon.

JAMES F. BALLARD, *Secretary.*

NEW ENGLAND OBSTETRICAL AND GYNECOLOGICAL SOCIETY

The fall meeting of the New England Obstetrical and Gynecological Society will be held on November 30, 1932, with headquarters at the University Club, Boston, Mass.

Clinics are to be held in the morning at the Massachusetts General Hospital and the Beth Israel Hospital with clinics in the afternoon at the Massachusetts Memorial Hospitals.

A luncheon will be served at the University Club at noon and a dinner at seven o'clock in the evening.

The speaker of the evening will be Dr. Barton Cook Hirst, eminent Philadelphian obstetrician and gynecologist.

FRED. J. LYNCH, M.D., *Recorder.*

SUFFOLK DISTRICT MEDICAL SOCIETY

The November meeting of the Suffolk District Medical Society will be a clinical meeting to be held at the Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, on Wednesday, November 30, 1932, at 8:15 P. M. The program will be presented by the Harvard Medical School and School of Public Health.

CLINICAL PROGRAM

1. How Emotions Disturb the Body. Dr. Walter B. Cannon.

2. Exophthalmic Goitre and Vitamin D Deficiency. Dr. Joseph C. Aub.

3. Field Studies in Common Colds. Dr. Wilson G. Smillie.

4. The Results of Serum Treatment in Pneumonia. Dr. Lloyd D. Felton.

WILLIAM H. ROBEY, M.D., *President,*
LELAND S. MCKITTRICK, M.D., *Secretary.*

BOSTON CITY HOSPITAL

Boston City Hospital Staff Clinical Meeting will be held Tuesday, November 22, 1932, at 8:15 P. M., in the Cheever Amphitheatre.

Dr. Joseph Resnik: Physiotherapy in Paralysis. 1. Hemiplegia. 2. Infantile Paralysis. 3. Peripheral Facial Paralysis. Discussion opened by Dr. Stanley Cobb, Dr. Joseph H. Shortell, Dr. Edward J. Monahan.

Dr. Otto J. Hermann: Physiotherapy in Fractures. Discussion opened by Dr. Waldo W. Robbins.

Dr. Girdie W. Dickinson, Dr. Richard P. Stetson: Presentation of Cases.

Dr. Stanley Cobb, Dr. Joseph Resnik: Report of experimental work on vasomotor changes produced by the galvanic current.

COMMITTEE ON HOSPITAL CLINICS.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

November 18—New England Roentgen Ray Society. See notice on page 862, issue of November 10.

November 21—Boston Medical History Club. See notice elsewhere on this page.

November 22—Boston City Hospital. See notice above.

November 30—New England Obstetrical and Gynecological Society. See notice elsewhere on this page.

December 7—American Board of Obstetrics and Gynecology. Announcement of Coming Examinations. See notice on page 557, issue of September 22.

December 7—New England Heart Association. See notice on page 813, issue of November 3.

December 9 - May 12—William Harvey Society. See page 765, issue of October 27.

December 12—Boston Orthopaedic Club. See page 812, issue of November 3.

February 6-10—The American College of Physicians. See page 765, issue of October 27.

February 17 and 18—The New England Hospital Association. See notice elsewhere on this page.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

January 4—Wednesday, Semi-Annual Meeting. Program to be announced.

May 3—Wednesday, Annual Meeting. Program to be announced.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Tentative Program
1932-1933

Wednesday, December 7, 1932—Beverly Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Robert Greenough. Subject: Postgraduate Instruction.

Wednesday, January 4, 1933—Danvers State Hospital, Hathorne. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Warren Stearns, Dean of Tufts College Medical School.

Wednesday, February 1, 1933—Council Meeting, Boston.

Wednesday, March 8, 1933—Lynn Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Soma Weiss, Boston City Hospital. Subject: Arterial Hypertension.

Wednesday, April 5, 1933—Essex Sanatorium, Middleton. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Wilson G. Smillie, Professor of Public Health Administration, School of Public Health, Harvard University. Subject: Common Colds.

Thursday, May 4, 1933—Censors' Meeting, Salem Hospital, 3:30 P. M.

Tuesday, May 9, 1933—Annual Meeting. Salem Country

Club, Forest Street, Peabody. Golf in afternoon; greens fee $2.00. Dinner at 7 P. M. Speaker: Dr. George Burgess Magrath. Subject to be announced later. Ladies invited.

R. E. STONE, M.D., Secretary.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The program of the Middlesex East District Medical Society for the coming year is as follows:

January 11 at Wakefield.

March 15 at Melrose.

May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, Secretary.

NORFOLK DISTRICT MEDICAL SOCIETY

November 29—Norwood Hospital, Norwood, Mass. Time to be announced. Papers by the Norwood Hospital Staff.

January 31, 1933—Roxbury Masonic Temple, 8 P. M. Subject: "Stomach Surgery"—Motion pictures. Dr. William R. Morrison.

February 28, 1933—Time and place to be announced. "The Diagnosis and Treatment of Anemia," by Dr. William P. Murphy.

March 28, 1933—Time, place and subject to be announced. Speaker will be Dr. J. P. O'Hare.

May—Annual Meeting. Time, place and subject to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline.

SUFFOLK DISTRICT MEDICAL SOCIETY

Wednesdays at 8:15 P. M.

November 30, 1932—Clinical Program, Peter Bent Brigham Hospital. See page 911.

January 25, 1933—General Meeting in association with the Boston Medical Library at the Boston Medical Library. Titles and speakers to be announced later.

March 29, 1933—Clinical Program, Massachusetts General Hospital.

April 26, 1933—Annual Meeting. Election of Officers. Scientific Program—Titles and speakers to be announced later.

The Medical Profession is cordially invited to attend all of these meetings.

WILLIAM H. ROBEY, M.D., President,
LELAND S. McKITTRICK, M.D., Secretary,
HILBERT F. DAY, M.D., Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

Schedule of meetings for 1932-1933:

Wednesday, December 14—Meeting at the Worcester Chamber of Commerce Auditorium. Paper by Dr. Joseph Meigs of Boston, entitled "Uterine Bleeding."

Wednesday, January 11—Dinner and meeting at the Worcester City Hospital. Paper by Dr. Morris Fishbein, Editor of the Journal of the American Medical Association.

Wednesday, February 8—Dinner and scientific meeting at the Worcester State Hospital. Program to be announced later.

Wednesday, March 8—Dinner and scientific program at the Memorial Hospital, Worcester. Program to be announced later.

Wednesday, April 12—Dinner and scientific meeting at the Rutland State Hospital. Scientific program to be announced later.

Wednesday, May 10—The Annual Meeting. Place and program to be announced later.

ERWIN C. MILLER, Secretary.

BOOK REVIEWS

Biochemistry in Internal Medicine. By MAX TRUMPER, PH.D., Clinical Chemist and Toxicologist; formerly in charge of the Laboratories of Biochemistry of the Jefferson Medical College and Hospital and of the Psycho-Biochemistry Laboratory, Graduate School of Medicine, University of Pennsylvania, Philadelphia, and ABRAHAM CANTAROW, M.D., Instructor in Medicine, Jefferson Medical College, Assistant Attending Physician, Philadelphia General Hospital; in charge of Laboratory of Biochemistry, Jefferson Hospital. With a foreword by Elmer H. Funk, M.D., Sutherland M. Prevost Professor of Therapeutics at Jefferson Medical College. 454 pages with illustrations. Philadelphia and London: W. B. Saunders Co., 1932. Cloth, $5.50 net.

This book is an interesting attempt to bridge the gap between the research laboratory and clinical medicine. It is written with an assumption that the reader knows something of the laboratory so that all the methods of complicated technic and discussion of values of methods are largely avoided. The variations of laboratory findings in various clinical conditions are well discussed and the physiological variations which are observed in the clinic are also well summarized.

In bridging this gap, however, the book loses somewhat in thoroughness. Thus, the interpretation of a laboratory finding may be stated, but the reasons why these interpretations are justified are not always adequately discussed. The value of a laboratory finding is largely dependent upon the method and technic used and this part of the problem is largely avoided.

One serious criticism of the book is that a bibliography has been omitted. References seem essential in a subject which is changing as rapidly as is the laboratory branch of clinical medicine. Thus, a statement of fact in 1932 would probably need to be qualified in a few years. A book which justifies its opinions by reference to original articles is, therefore, of far greater accuracy than one which lacks this essential detail.

For men who have some knowledge of the accuracy of laboratory procedures and who desire a summary of our present knowledge, this book fills a decided need.

Psychopathology of Forced Movements and the Oculogyric Crises of Lethargic Encephalitis. By SMITH ELY JELLIFFE, M.D., Ph.D. Nervous and Mental Disease Publishing Company, New York, 1932.

This book which is made up of material which appeared serially in the *Journal of Nervous and Mental Disease* is a critical review of the bibliography on the subject which is extraordinarily extensive and erudite, covering the subject seemingly from the time Eve lifted her eyes to the pendant apple in the garden of Eden to the latest ogling of the last Hollywood star. The author discards as inadequate and fallacious all the explanations, anatomic, physiologic, bacteriologic, chemical, etc., and advances the thesis that perhaps after all the phenomenon is merely a human reaction of the pattern extolled by the psalmist when he sang, "I will lift my eyes unto the hills." The reader marveling at the erudition of Dr. Jelliffe, who reminds one of Dr. Samuel L. Mitchell of old Philadelphia who was called a chaos of knowledge, is left wondering whether perhaps the learned doctor is not "spoofing".